A Dissertation on

STUDY OF QRS DURATION AND R/Q RATIO IN THE ASSESSMENT OF SEVERITY OF ACUTE MYOCARDIAL INFARCTION

submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

in fulfillment of the regulations

For the Award of the Degree of

M.D. (GENERAL MEDICINE)

BRANCH - I

KILPAUK MEDICAL COLLEGE

CHENNAI.

MARCH 2008

CERTIFICATE

This is to certify that "**STUDY OF QRS DURATION AND R/Q RATIO IN THE ASSESSMENT OF SEVERITY OF ACUTE MYOCARDIAL INFARCTION**" is bonafide work done by **Dr. SHYAM PADMANABHAN**, postgraduate student, Department of **Internal Medicine**, **Kilpauk Medical College**, Chennai-10 under my guidance and supervision in fulfillment of regulations of The Tamilnadu **Dr. M.G.R. Medical University** for the award of **M.D. Degree Branch I, Part II (General Medicine)** during the academic period from March 2005 to March 2008.

Dr. M. Dhanapal, M.D.,D. M.
The Dean
Kilpauk Medical College
Chennai 600 010

Prof. G. Rajendran, M.D.
Professor and Head
Department of Internal Medicine

Kilpauk Medical College

ACKNOWLEDGEMENT

I thank **Dr. M. Dhanapal, M.D., D. M.,** Dean, Kilpauk Medical College for permitting to use the resources and clinical material of this hospital.

I thank **Dr. G. Rajendran, M. D.,** Professor and Head of the Department of Internal Medicine for granting me permission to conduct this study.

I thank **Dr. Narayanaswamy Senguttuvan, M. D., D. M.,** Professor and Head of the Department of Cardiology for his valuable guidance and encouragement.

I am grateful to **Dr. Surendran E**. **M.D**., **Dr. Sundaramoothy. M.D.**, **Dr. Jayaprakash. M.D.**, **Dr. Manickam. M.D.**, Assistant Professors in the ICCU for providing immense help for the successful completion of this study.

I am grateful to **Dr. Mayilvahanan.S. M.D., Dr. Rohini I. M.D., Dr. Vasanthi.P. M.D.** for their encouragement, guidance and support during the course of the study.

I thank **Mr. Gopinathan.S.** for his valuable time spent in analyzing the data and providing statistical support.

I also thank my fellow Post Graduate students and house surgeons for all the timely help they rendered.

CONTENTS

INTRODUCTION

Within a short span of time, Ischaemic Heart Disease (IHD) will be the number one killer replacing infections, silently, slowly sometimes suddenly occluding the coronaries of millions of Indians. WHO study group on IHD and atherosclerosis described ischaemic heart disease as “cardiac disability, arising from reduction or arrest of blood supply to the myocardium in association with disease process in coronary arterial system.” During the past 30 years, a large decline in mortality due to coronary artery disease has been experienced in the West and there is a substantial increase in the developing countries. IHD accounted for 15.3 million deaths in 1996 which included 45.6% of all deaths in developed countries and 24.5% of all deaths in the developing countries. Estimated and projected rates of death (per l,00,000) due to this dreaded disease by World Bank Health Sectoral Priorities Review are 295 and 239 for males and females respectively in the year 2015.

Coronary artery disease has a multifactorial etiology with many of the risk factors being influenced by life style. Rapid change in dietary habits coupled with decreased physical activity in India as a consequence of urbanisation may partly explain the escalation of coronary artery disease. India is at going through a phase of rapid urbanization which has led to economic improvement, the consequence of which has resulted in fast food intake and tobacco consumption and decreased physical activity.

Atherosclerosis, which is the main cause of coronary artery disease is the accumulation of lipids, blood products, fibrous tissue, etc in the arterial wall. Although

the precise cause of atherosclerosis is unclear an emerging paradigm suggests that atherosclerosis involves multiple pathways in which lipoprotein entry and retention, injury to vessel wall from diverse stimuli and an associated long term inflammatory and immune response.

The standard 12 lead ECG has long been a reliable clinical tool for diagnosis of myocardial infarction. Minutes may be crucial in making the decision for urgent interventions in order to salvage the severely ischaemic myocardium. Besides history and physical findings the ECG may be the only clinical tool available readily in deciding to initiate life saving thrombolysis.

Identification of post infarction patients at risk for cardiac death and sudden cardiac death may lead to optimization of medical therapy and implantation of cardioverter-defibrillators. In the current era of technological development in cardiology there are a number of methods that could be utilised for the risk stratification purposes. Historically left ventricular ejection fraction was the first widely accepted risk stratifier in post infarction patients. Subsequently interest in Holter recorded ventricular arrhythmias, late potentials on signal averaged ECG, heart rate variability, substantiated prognostic value of these parameters in patients with prior myocardial infarction. New approaches, inc1uding heart rate turbulence, T wave alternans, QT variability and baroreflex sensitivity are being successfully tried for risk stratification purposes. Simultaneous1y invasive electrophysiology testing with induction of ventricular tachycardia or fibrillation has been utilized for risk stratification purposes. The concrete evidence for dire usefulness will known after extensive trials. All the above methods are worth exploring and there are some advantages and limitations of these methods. There is a limited

general access to the technology and methodology needed to implement some of these more sophisticated technique and also there are no standardizations of several of these methods. A standard 12 lead ECG is widely, easily available and serves as a powerful tool when considering risk stratification in post infarction patients. So a standard 12 lead ECG is a cost effective excellent tool for estimating the severity of myocardial infarction especially in developing countries like India.

AIMS AND OBJECTIVES

1. To study the “QRS duration” on short term prognosis of acute myocardial infarction.

2. To study the QRS duration in relation to 10-day hospital mortality.

3. To study R/Q ratio in lead II for the assessment of the severity of acute inferior wall myocardial infarction.

4. To study the R/Q ratio in lead II in relation to thrombolytic therapy in acute inferior wall myocardial infarction.

5. To study the comparison of R/Q ratio with QRS duration to assess the severity of acute inferior wall myocardial infarction.

REVIEW OF LITERATURE

At the threshold of the new millennium coronary artery disease is looming large as the new epidemic, afflicting Indians with severe and diffuse form of lesions. The prevalence of coronary artery disease increased in India during the latter half of the last century particularly among the urban population. (1)

The risk of coronary artery disease in Indians is 3 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese. Indians are prone as a community to coronary artery disease at a much younger age. (2)

In a metaanalysis Gupta and Gupta (1996) (3) estimated the prevalence of coronary artery disease among Indians to be 9.6% in the urban and 3.7% in the rural population. South Indians have a higher prevalence and 14 % in urban areas.

On screening persons over the age of 30 years by a 12 lead ECG in Chandigarh the prevalence was found to be 65.4 and 47.8 per 1000 males and females respectively. In a village of Haryana the prevalence was 22.8 and 17.3 per 1000 males and females respectively .(4)

Although the death rate from acute myocardial infarction has declined by about 30% over the last decade, it is seen as a fatal event at the onset itself is seen in approximately one third of patients. About 50% of the deaths associated with acute myocardial infarction occur within 1 hour of the event and are attributed to serious arrhythmia most often ventricular fibrillation. (5)

Almost all myocardial infarction results from coronary atherosclerosis, generally with superimposed coronary thrombosis, slowly forming high grade stenosis in epicardial coronaries may progress to complete occlusion, but yet does not usually precipitate acute myocardial infarction, probably because of the development of a rich collateral network over time. (6)

PATHOPHYSIOLOGY

On interruption of antegrade flow in an epicardial coronary artery, the zone of myocardium supplied by that vessel immediately losses its ability to shorten and perform contractile work (7), Four abnormal contraction patterns develop in sequences

a. Dyssynchrony - Dissociation in the time course of contraction of adjacent segments.

b. Hypokinesis - Reduction in the extent of shortening.

c. Akinesis - Cessation of shortening.

d. Dyskinesis - Paradoxical expansion and systolic bulging.

Accompanying the dysfunction of the bulging segment, initially there is hyperkinesis of the remaining normal myocardium. This increased motion subsides within 2 weeks of infarction, during which time, some recovery can be seen in the infarct region as well. Patients with acute myocardial infarction also show reduced myocardial function in the non-infarcted lesion i.e. ischemia at distance.(8)

Clinical features: Chest pain and chest discomfort, are the predominant presenting features of myocardial infarction. Nausea, vomiting and epigastric discomfort, stimulating an abdominal pathology, occurs more commonly in patients with IWMI than AWMI. The stimulation of vagus nerve or Bezold-Jarisch reflex is presumed to be the mechanism involved. In elderly patients and diabetics, acute myocardial infarction can manifest clinically without chest pain, but with symptoms of acute LVF and chest tightness, or by marked weakness, or syncope. (9) Patients usually appear anxious and in considerable distress. Sinus bradycardia is particularly frequent in, patients with inferior and posterior wall infarction. (10) Hypotension, engorged neck veins, clear lung fields and heart blocks are characteristic features of right ventricular myocardial infarction, which occurs in about one-third cases of IWMI. (11)

On cardiac auscultation a fourth heart sound is almost always present in, patients with acute myocardial infarction in sinus rhythm and this is usually best heard between the left sternal border and the apex. In cases of RVMI associated with papillary muscle dysfunction of the tricuspid valve, murmur of TR may be present. (7) In, patients with IWMI, posterior involvement are associated with development of significant MR (12).

The incidence of cardiogenic shock and severe LVF occurs less frequently in IWMI, unless otherwise, it is complicated by mechanical defects such as ventricular septal rupture, papillary muscle dysfunction or predominant RV infarction (7).

Cardiac Enzymes

Estimation of the rise and fall in serum cardiac markers is one of the WHO criteria for diagnosis of AMI, other being history of ischemia and electrocardiographic changes. (13).

In myocardial infarction, as myocytes become necrotic, the integrity of the sarcolemmal membrane is compromised and intracellular macromolecules (serum cardiac markers) begin to diffuse into the cardiac interstitium and ultimately into the microvasculature and lymphatics in the region of infarcts. (14) The rate of appearance of these macromolecules depends on several factors including intracellular location, molecular weight, local blood supply and lymphatic flow, and the rate of elimination from the blood. (15).

Among the various serum cardiac markers; cardiac specific troponin (Tn-c, Tn-I), Creatinine kinase (CPK-MB), Myoglobin and LDH are the commonly measured, with troponin now considered as the preferred biomarker for diagnosis of acute myocardial infarction (16). Serum level of cardiac enzymes appears to be the most practical means of estimating infarct size (17). In addition, cardiac troponin measurements have been shown to have prognostic value for identifying patients with an acute coronary syndrome at risk for adverse clinical outcomes and who also exhibit enhanced responsiveness to new therapies such as glycoprotein IIb/IIIa inhibitors and low molecular weight heparin (18).

Studies have shown that patients with IWMI who have anterior ST segment depression and RVMI, have got higher cardiac enzyme values, indicating a larger mass of myocardial involvement. (19)

ELECTROCARDIOGRAPHY

The value of ECG in diagnosing and localizing the size of infarction is unequivocal. (20) The 12 lead ECG remains the centre of decision pathway for management of patients with acute coronary syndrome and to distinguish between the presentations of those with and without ST segment elevation (21). Katz et al (1946)(22) set the criteria for electrocardiographic diagnosis of myocardial infarction. Myocardial infarction was classified into transmural and non-transmural. Transmural were those demonstrating significant Q wave plus typical ST-T wave alteration and T wave inversion. In non-transmural infarcts, the established ST segment alteration and T wave inversion persisted beyond 7 days, but with no significant Q waves.

The inferior wall of the left ventricle is directed to the standard leads II, III and aVF (23). The hyperacute phase is manifested by increased ventricular activation time, increased amplitude of R wave, straightening and subsequent slope elevation of the ST segment, tall and widened T waves.

In the fully evolved phase, standard lead III commonly reflects a QS complex, while standard lead II and aVF however usually reflect a QR complex. The disappearance of the small normal initial Q in lateral leads is a corroborative sign of IWMI. Reciprocal ST segment depression usually occurs in the right precordial leads. (24)

Q waves are frequently seen in lead III in normal patients, so to enhance the specificity of Q wave in lead III, guidelines have been laid down. According to Harpaz D et al (1999) (25), Q wave should he greater than 0.03 seconds. To increase the specificity of Q wave in lead III, Minnesota code modification (26) advised:

- Q in Lead II greater than 0.0 3second.
- Lead III Q greater than 0.03sec and Q greater than or equal to 1mm in lead aVF.
- Lead aVF, Q greater than 0.03 see.

Along with IWMI, changes in the lateral leads (l, aVL, V5, V6) with an isoelectric or elevated ST segment in lead I identifies obstruction of the circumflex coronary arteries with a sensitivity specificity and predictive value of 83%, 96% and 93% respectively. Changes in lateral leads are rare in IWMI resulting from obstruction of the RCA. (27)

ECHOCARDIOGRAPHY

Evaluation of LV Function:

The parameters of systolic and diastolic function can be obtained by M mode or 2D echocardiography, either by measuring left ventricular dimension in systole and diastole or with LV volumes calculated by area length method in apical2D view.

Systolic Function: The three commonly used indices of systolic LV function are

LV Ejection Fraction = (EDV - E3V / EDV) X 100

Normal- in male 59±6 and in female 58±7

Fractional Shortening (FS) = (LVIDd - LVIDs / LVIDd) X 100

Normal - > 24 %

E-Point Septal Separation (EPSS)

Normal - Upto 8 mm.

Diastolic Function: The function is assessed by Doppler echocardiography by calculating several indices but those in common use include peak ‘E’ velocity, peak ‘A’ velocity, E/A ratio and isovolumeic relaxation time. (28)

Segmental Wall Motion Analysis

The American Society of Echocardiography Committee (ASE) on Standards recommended a semi-quantitative method that derives wall motion score based on a visual impression of regional wall motion. The left ventricular mass can be divided into 3 equal levels from the apex to base length, resulting in its partition into basal, middle and apical levels. They proposed a 16-segment model for visual semi-quantitative wall motion analysis. (29)

The proposed segments are

Base	Mid	Apical
Basal Anterior	Anterior	Anterior
Basal Anteroseptal	Anteroseptal	Inferior
Basal lnferioseptal	Inferioseptal	Lateral
Basal Inferior	Inferior	Septal
Basal Posterior	Posterior	
Basal Lateral	Lateral	

The five basic wall region visualized are

Anterior Wall: Which was considered to extend over the anterior surface of ventricles from the anterior interventricular sulcus, around the free ventricular wall to the origin of the anterior papillary muscle.

Posterolateral wall: was considered to extend posteriorly between the papillary muscles.

Inferior wall: Extended from the posterior papillary muscle to the junction with the septum at the posterior interventricular sulcus.

Septal Region: Included the septum.

Apical Region: Included the very tip of ventricular cavity and apex.

One can relate the various segments to coronary artery distribution

Anterior and anteroseptal segments in both basal and middle third and the apical segments - left anterior descending artery distribution.

Basal, lateral and middle lateral - left circumflex artery distribution.

Posterior and inferoseptal in both the basal and middle third - right coronary artery distribution.

Scoring Scale: Henry et al (1979) (30) stated that at least 50% of the endocardium must be visualized in anyone segment throughout the cardiac cycle for reliable prediction of presence or absence of LV and RV asynergy. A significant concern was the ability to grade 2D echo images and distinguish normal from hypokinetic wall

motion. For this, strict attention was paid not only to the endocardial inward motion but also to the wall thickening. Liberman et al (1981) (31), showed that systolic thickening provided better separation of normal from infarcted myocardium than endocardial motion alone.

The ASE committee has also proposed the following scoring scale for standardization of wall segment motion

Score	Wall Motion	Definition
1.	Normal	Normal endocardial inward motion and wall thickening in systole
2.	Hypokinesis	Reduced endocardial motion and wall thickenil1g in systole
3.	Akinesis	Absence of endocardial inward motion or wall thickening in systole
4.	Dyskinesis	Outward motion or bulging of the segment in systole, usually associated with thin, scarred myocardium.

Wall Motion Score Index: It can be derived from the sum of all scores divided by the number of segments visualized. (32).

Assessment of Overall Performance of the Ischemic Left Ventricle:

Left ventricular ejection fraction (LVEF) is one of the most commonly used indices of systolic LV function. 2DE is a useful noninvasive method for estimation of LV volume and LVEF.

Following myocardial infarction, there is a decrease in LVEF and this is directly related to the amount of damaged myocardium and the extent of potentially ischemic muscle. Morbidity and mortality rates correlated well with initial ejection fraction. Patients with ejection fraction <30-35% had greater risk of pump failure and death. Patients with AWMI had a lesser LVEF (38±14) as compared to those with inferoposterior wall myocardial infarction (55±10) because a greater region of myocardium was involved in AWMI. (33).

In patients with IWMI, both LVEF and RVEF are depressed, whereas in patients with AWMI, RVEF generally remains normal. But while RV performance rapidly improves with prompt return to normal level, as early as 2 days after infarction, there is less improvement in the LVEF. (34)

Carr et al (1979) (35) in their study found that cross-sectional echocardiography and radionudeotide angiography were of equal value in the estimation of LVEF. Although radionuc1eotide ventriculography generally provides a more exact measurement of ejection fraction, echocardiography appears to be capable of directly or subjectively evaluating ventricular function well enough to identify patients with risk of death from myocardial infarction. (33).

COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

Mechanical Complications:

1. Left Ventricular Failure: Even in thrombolytic era, left ventricular dysfunction remains the single most important predictor of mortality after acute myocardial infarction. In patients with acute myocardial infarction, heart failure is characterized either by systolic dysfunction alone or by both systolic and diastolic dysfunction. Left ventricular diastolic dysfunction leads to pulmonary venous hypertension and pulmonary congestion whereas systolic dysfunction is principally responsible for a depression of cardiac Output and of the ejection fraction. Clinical manifestations of left ventricular failure become more common as the extent of the injury to the left ventricle increases; mortality increases in association with the severity of the hemodynamic deficit.

Hemodynamic Classification of Patients with Acute Myocardial Infarction: **(Killip Classification)**

Class	Based on Clinical Examination	Based on Invasive Monitoring
I	No evidence of pulmonary congestion	Normal
II	Mild evidence of pulmonary congestion	Pulmonary congestion PCWP > 18 CI<2.2
III	Pulmonary oedema	Peripheral Hypoperfusion PCWP>18 CI>2.2
IV	Cardiogenic Shock	Pulmonary congestion and peripheral hypoperfusion PCWP>18,CI>2.2

2. **Cardiogenic Shock**: The severest clinical expression of left ventricular failure is associated with extensive damage to the LV myocardium (about 40%) in more than 80% of acute myocardial infarction patients in whom it occurs. The remainder have a mechanical defects such as ventricular septal defect or papillary muscle rupture or predominant right ventricular infarction. In the part cardiogenic shock has been reported to occur in upto 20% of parents with acute myocardial infarction, but estimates from recent large randomised trials of thrombolytic therapy and observational databases report an incidence rate in the range 7% About 10% of patients with cardiogenic shock present with this condition at the time of admission, whereas 90% develop it during hospitalisation (36). Patients with cardiogenic shock due to acute myocardial infarction are more likely to be older to have a history of a prior myocardial infarction or congestive heart failure and to have sustained an anterior infarction at the time of development of shock.

Arrhythmias:

Electrical instability: VPC's, VT, VF, AIVR, NPAVJT

Pump failure/ excessive sympathetic stimulation: Sinus tachycardia AF / Afl, PSVT

Bradyarrhythmias and conduction disturbances

Sinus bradycardia, junctional escape rhythm AV block and intraventricular block

Progression of Acute Myocardial Infarction: Most of the thrombolytic trial shows that mortality in IWMI is about half that of AWMI.

Name of Studies	AWMI			IWMI		
	No. of Deaths	No. of Patients	%	No. of Deaths	No. of Patients	%
GISSI-I	403	2193	18.4	145	2004	7.2
ISIS	329	1827	18.0	185	2112	8.8
AIMS	51	292	17.5	26	225	7.8
ASSRT	134	796	16.8	77	773	10.0
LATE	58	410	14.2	30	351	8.6
	Average - 16.98%			Average – 8.48%		

Certain high risk subgroups can be identified in IWMI by single ECG criteria. These are right precordial ST segment elevation, left precordial ST segment depression and high degree AV block. 2D echo can also diagnose high risk subgroups like those having RV and posterior wall assynergy, patients having poor ejection fraction, high wall motion index score, VSD, papillary muscle ruptures etc. These high risk subgroups have similar mortality rates as of AWMI, while the low risk subgroups have got mortality rates around 2-4 % (37).

The results of thrombolytic trials in myocardial infarction show that thrombolysis in IWMI is statistically not as significant as in anterior wall myocardial infarction and it has got only a meager impact in the mortality reduction.

Studies		Thrombolytic Deaths/ Patients	Control Deaths/ Patients
Streptokinase	GISSI-I (0-12 h)	137/2009 (6.8%)	145/2004 (7.2%)
	ISIS-2 (0-24h)	150/2076 (7.2%)	185/2112 (8.8%)
SK/ASA	ISIS-2 (0-24h)	69/1016 (6.8%)	107/1047 (10.2%)
Altepase	ASSET (0-5h)	46/734 (6.3%)	77/773 (10.0%)
	LATE (6-12h)	34/381 (8.9%)	30/351 (8.6%)

Prognostic Significance of ECG in Acute Myocardial Infarction

Standard 12 lead ECG serves as an extremely useful tool in risk stratification after myocardial infarction. Careful analysis of ECG provides comprehensive information about pathology of the heart, which could lead to cardiac events including reinfarction, progression of heart failure and death. Identification of post infarction patients at risk or cardiac death and sudden cardiac death may lead to optimization of medical therapy and implantation of cardioverter defibillators.

QRS Duration and Myocardial Infarction

In a study by Pudil et al (2001) (38), acute myocardial infarction with intermediate QRS duration (0.09-0.11sec) on admission electrocardiogram was independently associated with increased 7 day, 0 day and 1 year all cause mortality.

More recently it was shown that QRS duration of the admission electrocardiogram is independently associated with 30 days and 1 year mortality after acute myocardial infarction in the GUSTO-1 trial. In this trial QRS prolongation had a more significant outcome in patients with anterior myocardial infarction (39).

Prolonged QRS duration on surface electrocardiogram was associated with left ventricular dysfunction in patients referred to radionucleotide exercise ventriculography. It was also reported that QRS (duration > 0.1 seconds indicated decreased left ventricular ejection fraction .(40).

A QRS of < 0.09 seconds on admission electrocardiogram is indicate of a relative benign outcome compared with a QRS duration of >0.09 seconds.

The mechanism by which prolonged QRS duration on admission is associated with increased risk of death is not clear. Multiple physiologic parameters associated with poor outcome can lead to prolonged QRS duration in patients with acute myocardial infarction including increased left ventricular muscle mass, myocardial fibrosis, increased area of necrosis, involvement of conduction system in the ischaemic area, poor metabolic state that slows conduction, and effects of various medications.

After adjusting for all significant variables associated with mortality, including age, gender, diabetes mellitus, smoking, systemic hypertension, Killip class>2 on admission and anterior location of myocardial infarction prolonged QRS duration both in the those with QRS duration> 0.11 seconds was found to be independently associated with increased 7 day, 30 day and 1 year mortality (38).

QRS prolongation was positively associated with older age, female gender, anterior myocardial infarction and congestive heart failure on admission.

In 743 patients of the placebo arm of the cardiac arrhythmia suppression trial 1991 with stable coronary artery disease and QRS duration >100 ms, the risk ratio was 1.4 for new or worsening congestive heart failure, 1.5 for arrhythmic death or cardiac arrest and 1.4 for all cause mortality ($p<.05$). In post acute myocardial infarction patients QRS prolongation was significantly correlated with arrhythmic events (41).

In patients with normal coronary arteries, QRS duration decreases with exercise probably because of an increase in the sympathetic tone. In contrast, in patients with coronary artery disease, QRS duration increases during exercise testing. Michaelids et al, 1993(42) reported that exercise induced QRS prolongation was proportional to the number of coronary arteries with >70% stenosis. Mean QRS prolongation was 4.8±7.5 ms in patients with I-vessel disease, 7.8±11.8 ms in patients with 2-vessel disease and 13.3±12.1 ms in patients with 3-vessel disease ($p< 0.001$).

Distortion of the terminal portion of the QRS complex in the admission ECG is an independent predictor of increased hospital mortality in patients receiving thrombolytic therapy >2 hour after the onset of symptoms. During regional myocardial ischemia the conduction velocity of the activation wave in the Purkinje fibres is prolonged .(43)

QRS prolongation on surface electrocardiography has been identified as a marker for increased cardiac mortality. A potential mechanism for increased mortality is ventricular tachycardia. Patients with prolonged QRS duration were older had lower LVEFs and were more likely to have a history of myocardial infarction. Prolonged QRS

was a significant predictor of sustained monomorphic VT inducibility ($p< 0.0001$). On Multivariate analysis correcting for age, sex, LVEF, history of myocardial infarction, medication and QRS conduction delay proved to be independently associated with sustained monomorphic VT inducibility (relative risk 3.290, 95% confidence interval 2.185 to 4.953 for prolonged vs. normal QRS duration) (44).

At multivariate analysis, prolonged filtered QRS duration had an independent relation to late arrhythmic events after acute myocardial infarction. (45)

QRS duration remains a very powerful predictor of future cardiac events in post infarction patients (46). QRS duration reflects well the magnitude of left ventricular dysfunction and therefore not surprisingly is a powerful predictor of mortality in post infarction patients.(40). A study by Fadl et al (2003) (47) in a large population of post infarction patients indicate that QRS duration 0.12 second is associated with hazard ratio of 1.7; $p = 0.001$.

Significance of R/Q Ratio in Lead II in Inferior Wall Myocardial Infarction

Inferior wall of the heart is constituted by the inferior wall of left ventricle and is oriented to the positive electrodes of lead II, III, avF.

Inferior wall myocardial infarction has 3 phases

1. Early hyper acute - characterized by elevation of ST segments.

2. Ful1y evolved phase - characterized by the presence of pathological Q or QS complexes coved and elevated ST segments and inverted sharply pointed and symmetrical T waves in lead II, III, avF.

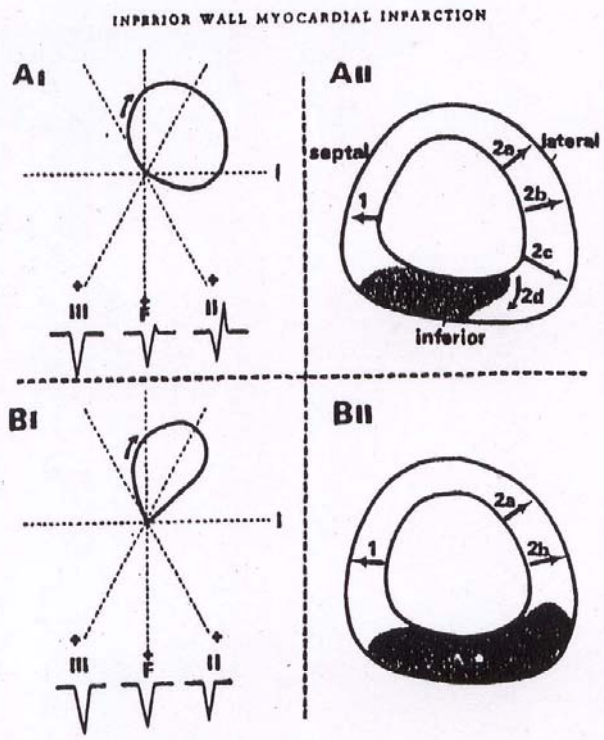
3. Chronic stabilized phase - Characterized by residual Q wave abnormalities in leads II, III, avF, particularly in lead II, avF.

Pathological Q waves of inferior wall myocardial infarction are not usually deep or as wide as the pathological Q waves which occur with anterior wall myocardial infarction because inferior wall myocardial infarction is reflected by extremity leads (48).

In IWMI a QS complex is usually present in lead III and avF. Standard lead II usually reflects Qr or QR complex. In inferior wall myocardial infarction, among the inferior leads tallest terminal R wave is seen in lead II. (48)

We can explain this distribution of terminal R wave by considering the anatomy of inferior wall. We can divide the inferior wall into right lateral and left lateral region. Lead III is oriented to right lateral region and lead II to left lateral region.

The brunt of infarction affects the right lateral region of inferior wall with sparing of left lateral region so lead III reflects the largest and widest QS complex, lead II however is oriented to left lateral region of inferior wall reflects the potentials of healthy overlying muscle as a terminal R wave; so loss of R wave in lead occurs mainly in extensive inferior wall myocardial infarction



A is a diagramatic representation of the frontal plane vector loop in inferior wall myocardial infarction with sparing of the inferolateral wall B, is a diagramatic representation of the frontal plane vector loop in extensive inferior wall myocardial

Two major determinants of clinical outcome in patients with acute myocardial infarction are the extent of infarction and the residual left ventricular function (49).

In patients with inferior wall myocardial infarction terminal R wave and R/Q ratio in lead II reflects the extent of infarction and residual function so by calculating R/Q ratio in lead II we can define the severity of inferior wall myocardial infarction.

Alexander Arditti et al (1985) (50) studied a simplified QRS scoring system for the estimation of the severity of acute inferior myocardial infarction. Electrocardiographic assessment of the R/Q ratio in lead II of patients with first acute inferior wall myocardial infarction offers important indirect evidence of severity and extent of myocardial infarction. An R/Q ratio of more than 2 predicts mild and localized left ventricular involvement with good global left ventricular function and good clinical course. An R/Q ratio between 1 and 2 predicts a greater degree of local asynergy with some lateral extension with reduced global left ventricular function, but still a good clinical outcome. R/Q ratio less than one predicts severe inferior wall asynergy with high incidence of lateral wall involved, reduced global left ventricular function and complicated clinical course during the acute phase.

Eliezer et al (1988) (51) conducted similar study in which acute inferior wall myocardial infarction were divided into three groups according to the R/Q ratio in lead II. This was done to correlate these groups with characteristic course to electrocardiographic status. Patients with R/Q > 2 (group 1) had a more rapid progression through the electrocardiographic stages along with better clinical course than patients with lower R/Q ratio. Patients in group In with R/Q < 1 had a slower electrocardiographic stage progression which correlates well with a more complicated clinical course. Group II was an intermediate group in both the electrocardiographic and clinical course. It is suggested that the R/Q ratio in lead II can be used as a marker of the severity of IWMI since it correlates well with the course of electrocardiographic stages. This may be an additional, simple and inexpensive electrocardiographic tool for following the natural course of IWMI.

Lewin (1986) (52) conducted a study in which acute inferior wall myocardial infarction were divided into three groups. Group I predominant right ventricular infarction, group II combined right and left ventricular infarction and in group III predominant left ventricular infarction. Patients with predominant RV infarction (Group I) had smaller Q wave values and taller mean R wave in the inferior leads than patients in the other groups. In predominant RV infarction R/Q ratio> 2.5 and is combined RV and LV or extensive LV infarction R/Q ratio is < 2.5.

MATERIAL AND METHODS

The study was carried out on 75 patients admitted in ICCU of Department of Medicine, Government Royapettah Hospital, Kilpauk Medical College, Chennai-10.

SELECTION CRITERIA:

In this study 75 cases of both sexes of more than 20 years of age with first episode of myocardial infarction, typical chest pain of more than 30 minutes, onset of symptoms within previous 6 hours, at least 0.2 mv ST segment elevation in two or more contiguous precordial leads or at least 0.1mv ST segment elevation in two or more leads were included.

The screening criteria for the identification of the presence of myocardial infarction were:

- Inferior criterion: Q>30 msec in lead aVF
- Anterior criteria: A Q or R< 0.1 mv and < 10 m sec in V2.

EXCLUSION CRITERIA

Patients with ECGs showing evidence of LBBB, RBBB, LAHB, LVH, old myocardial infarction, preexicitation syndrome were excluded from the present study.

All cases were subjected to following examination.

Case number, registration number, father/husband name, age, sex, socio-economic status and rural urban background etc.

Presenting complaints with duration were noted in detail such as chest pain, its site, duration and radiation, accompanying features like sweating, nausea, vomiting, syncope, breathlessness, palpitation, oedema, abdominal distension, right hypochondrial pain and other atypical symptoms.

Detailed symptomatology of clinical presentation was sought with special reference to onset, duration, intensity, relationship to circadian and seasonal rhythm of symptoms.

Past History

It was taken to exclude the presence of a previous myocardial infarction and to find out the risk factors including hypertension, diabetes mellitus and other evidence of atherosclerotic disease like CVA.

Family History

Family history of diabetes mellitus, coronary artery disease, hypertension, dyslipidemia, age of death were enquired for.

Personal History

It was taken with special reference to addictions like tobacco chewing, smoking, alcohol consumption, dietary habits etc.

General Examination:

It was done meticulously in all cases, special care was taken to record pulse rate, rhythm, volume, character and condition of arterial wall. Blood pressure was taken to supine position. Presence of pallor, cyanosis, pedal oedema and raised JVP was noted.

Systemic Examination

- Detailed Cardiovascular system examination was done by observing, palpating apex impulse, thrills, precordial pulsations were looked for, heart sounds S1, S2, S3 and S4, murmurs were auscultated. (site, duration, timing, character and radiation).
- Respiratory system was examined by auscultating for breath sounds and to look for evidence of pulmonary oedema.
- Abdominal examination was done for the presence of any organomegaly and ascites.
- Central nervous system was examined for presence of any neurological deficit.

Investigations

The following investigations were done in all cases.

- Haemogram - Hb, TLC, DLC, ESR.
- Urine - Albumin, Sugar and Microscopy
- Blood - Urea, sugar, Serum cholesterol and serum creatinine.

Electrocardiogram:

12 lead ECG was taken in all cases taking special precautions while placing the chest leads. Repeat ECGs were taken every day on first three consecutive days of admission followed by every alternate day until 10th day.

Rigorous Definition of Wave Forms

When the initial aspect of the QRS complex is negative, a Q wave is present. Prior to returning to the baseline, this negative deflection may be smooth or notched. A smooth q wave is present when the initial negative deflection contains no reversal in the direction of 0.05 mv or more.

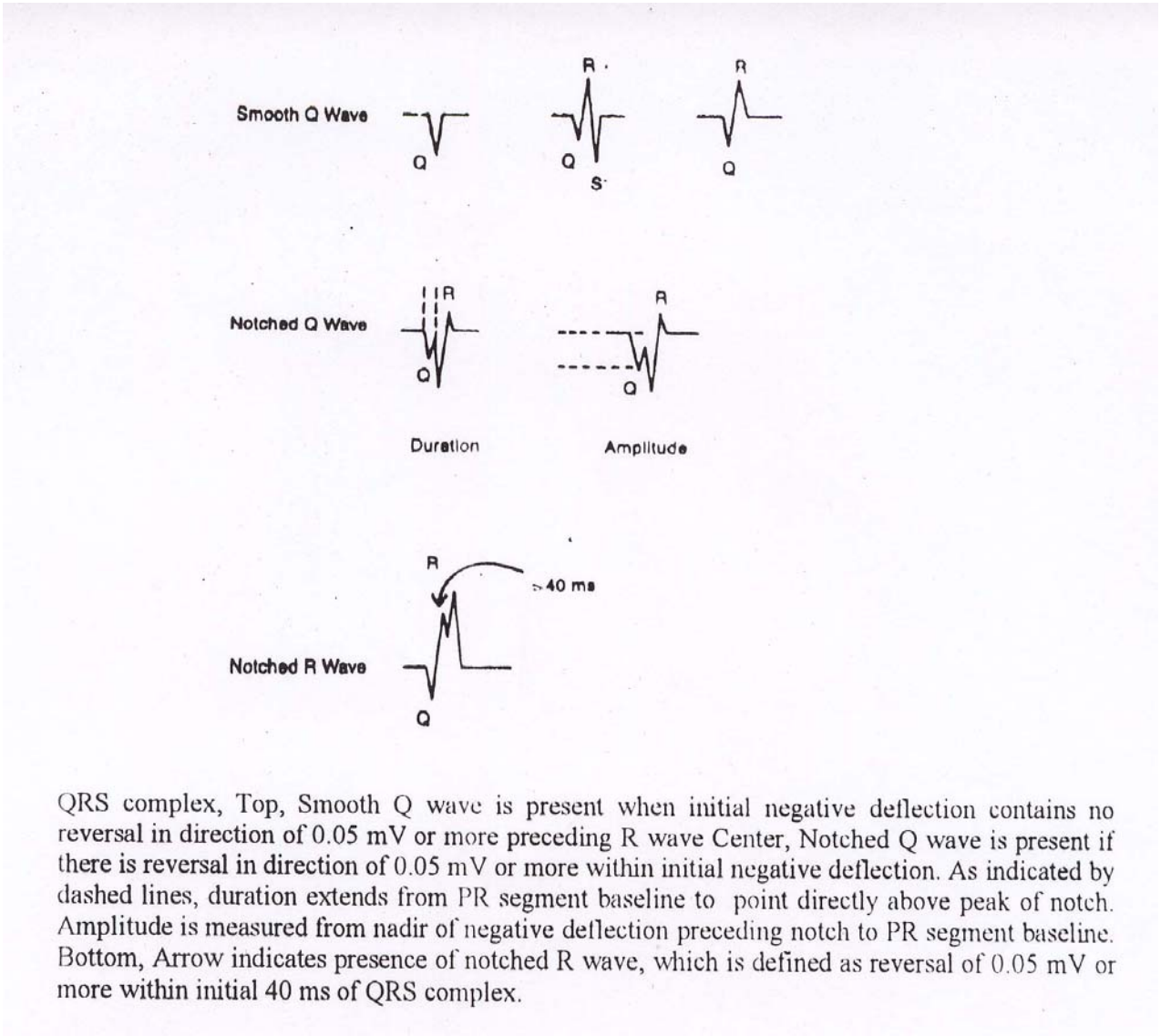
The duration and amplitude of such Q wave are measured as the width and depth respectively of the initial negative deflection.

A notched Q wave is present if there is a reversal in direction of 0.05 mv or more within the initial negative deflection. In this instance, the duration of the Q wave should be measured along the PR baseline only to the point directly above the peak of the notch and remainder of the negative deflection should not be considered. The amplitude of the Q should be measured to the nadir of the negative deflection preceding the notch.

An R wave is defined on the initial positive deflection. A notched R wave is present if there is a reversal in direction of 0.05 mv or more within the initial 40 ms of the QRS complex.

Accurate Wave from Measurement:

Careful manual measurement of both amplitudes and duration should be made with calipers using center of the trace of the inscribed waveform.



QRS complex, Top, Smooth Q wave is present when initial negative deflection contains no reversal in direction of 0.05 mV or more preceding R wave Center, Notched Q wave is present if there is reversal in direction of 0.05 mV or more within initial negative deflection. As indicated by dashed lines, duration extends from PR segment baseline to point directly above peak of notch. Amplitude is measured from nadir of negative deflection preceding notch to PR segment baseline. Bottom, Arrow indicates presence of notched R wave, which is defined as reversal of 0.05 mV or more within initial 40 ms of QRS complex.

Rand Q wave amplitude and resultant R/Q ratio were calculated in lead-II using the mean value of 3 consecutive QRS complexes and the TP segment as baseline on 3rd

and 7th day of hospitalisation. By using 3rd day R/Q ratio on lead-II short term prognosis of inferior wall myocardial infarction was evaluated. By comparing R/Q ratio on 7th and 3rd day the relationship of thrombolysis and R/Q ratio was evaluated.

For the study, patients of inferior wall myocardial infarction were divided into groups according to R/Q ratio in lead II on 3rd day.

Group I	Includes patients of IWMI with R/Q ratio> 2
Group II	Includes patients of IWMI with R/Q ratio 1- 2
Group III	Includes patients of IWMI with R/Q ratio < 1

For the study of QRS duration, widest QRS duration in standard lead was manually measured on 3rd day of hospitalisation.

Only leads without extreme ST segment deviation were considered. According to the QRS duration, patients of myocardial infarction including both inferior and anterior wall myocardial infarctions were divided into 3 groups.

Group A	With QRS duration < 0.09 See.
Group B	With QRS duration 0.09 - 0.11 sec (intermediate QRS prolongation)
Group C	With QRS duration > 0.11 sec (Significant QRS prolongation)

ECHOCARDIOGRAPHY

The 2 dimensional echocardiography study was performed on the 7th day post myocardial infarction, with the patient in the left lateral decubitus position with Toshiba Model SSH -140 echocardiography machine. A 3.75 MHz phased array sector transducer was used. The left ventricle was studied as recommended by American Society of Echocardiography in the long and short view.

The committee proposed the following scoring scale for standardization of wall segment motion.

A normally contracting segment or a hypercontracting segment is assigned a score of 1, Hypokinesia 2, Akinesia 3, Dyskinesia 4 and aneurysmal segment 5.

Dyssynergy of a LV segment was defined as Hypokinesis, Akinesis or Dyskinesis involving> 50% of that segment.

The ejection fraction was calculated by the standard formula as recommended by American Society of Echocardiography 1989(ASE).

Left Ventricular Ejection Fraction:

$$(LVEF) = \frac{EDV - ESV}{EDV} \times 100$$

EDV = End Diastolic volume

ESV= End Systolic Volume

Normal> 60%

The QRS duration and R/Q ratio were evaluated with ejection fraction and regional wall motion abnormalities.

Statistical Analysis

In present study comparison among the three groups using tables and graphic presentations were performed. The analysis between groups was performed using student ‘t’ test or the Chi Square test when indicated. All data were expressed as mean standard deviation. A value of < 0.05 was considered statistically significant.

OBSERVATION

The present series “Study of QRS Duration and R/Q ratio in the assessment of Severity of Acute Myocardial Infarction” was carried out on 75 patients admitted in ICCU of Department of Medicine, Government Royapettah Hospital, Kilpauk Medical College, Chennai-10.

Table No.1

Distribution of Type of Myocardial Infarction

S. No.	Type of Myocardial Infarction	No. of Cases	Percentage
1.	AWMI	28	37.33
2.	IWMI	44	58.67
3.	AWMI+ IWMI	3	4.0
Total		75	100.0

58.67% of the patients were having inferior wall myocardial infarction, 37.33% were having anterior wall myocardial infarction while 4.0% patients had both anterior and inferior wall myocardial infarction.

Table No. 2

Thrombolytic Therapy in Acute Myocardial Infarction

S. No	Type of M.I.		Total	Thrombolyzed		Non Thrombolyzed	
		No.	%	No.	%	No.	%
1.	AWMI	28	37.33	14	50.0	14	50.0
2.	IWMI	44	58.67	24	54.54	20	45.46
3.	AWMI +IWMI	3	4.00	2	66.67	1	33.33
	Total	75	100.0	40	53.33	35	46.67

$X^2 = 6.06$, $p < 0.05$ Significant

Out of total cases 53.3% patients were thrombolyzed.

Table No. 3

Distribution of Ejection Fraction

Type of Myocardial Infarction	< 40%		41-50 %		> 50%	
	No.	%	No.	%	No.	%
AWMI (n-26)	9	34.61	13	50.0	4	15.38
IWMI (n-41)	7	'17.07	21	51.21	13	31.70
AWMI +IWMI (n-3)	1	33.33	2	66.67	-	-
Total (n-70)	17	24.28	36	51.42	17	24.28

X2 = 41.86, $p < 0.001$ Highly Significant

Five patients expired before Echocardiogrnphy

Most of the patients with of anterior wall myocardial infarction had left ventricular ejection fraction in the range of 41-50%. Nine patients (34.61%) with anterior wall myocardial infarction had left ventricular ejection fraction < 40%, against 17.07% patients of inferior wall myocardial infarction.

Table No. 4

Distribution of Myocardial Infarction in Relation to QRS Duration

ORS Duration (In Seconds)	IWMI		AWMI		Total
	No.	%	No.	%	
Group A (<0.09)	32	65.30	17	34.69	49
Group B (0.09- 0.11)	9	50.0	9	50.0	18
Group C (>0.11)	6	75.0	2	25.0	8
Total	47	62.67	28	37.33	75

$X^2 = 13.70$, $p < 0.05$ Significant

65% of the patients with combined inferior wall myocardial infarction and anterior wall myocardial infarction were distributed in group A (<0.09) 40% patients with anterior wall myocardial infarction alone had QRS duration >0.09 seconds as compared to 30% patients of inferior wall myocardial infarction.

Table No. 5

QRS Duration and Cardiac Arrhythmias in Myocardial Infarction

Arrhythmia	Group A (< 0.09) (n-49)		Group B (0.09 – 0.11) (n-18)		Group C (> 0.11) (n-8)		Total	
	No.	%	No.	%	No.	%	No.	%
S.V.E.	3	50.0	1	16.67	2	33.3	6	16.21
V.E.	12	66.67	4	22.22	2	11.11	1R	48.04
S.V.T.	-	-	2	10O.n	-	-	2	5.4
V.T.	3	33.33	4	44.44	2	22.22	9	25.0
C.H.B.	-	-	2	100.0	-	-	2	5.4

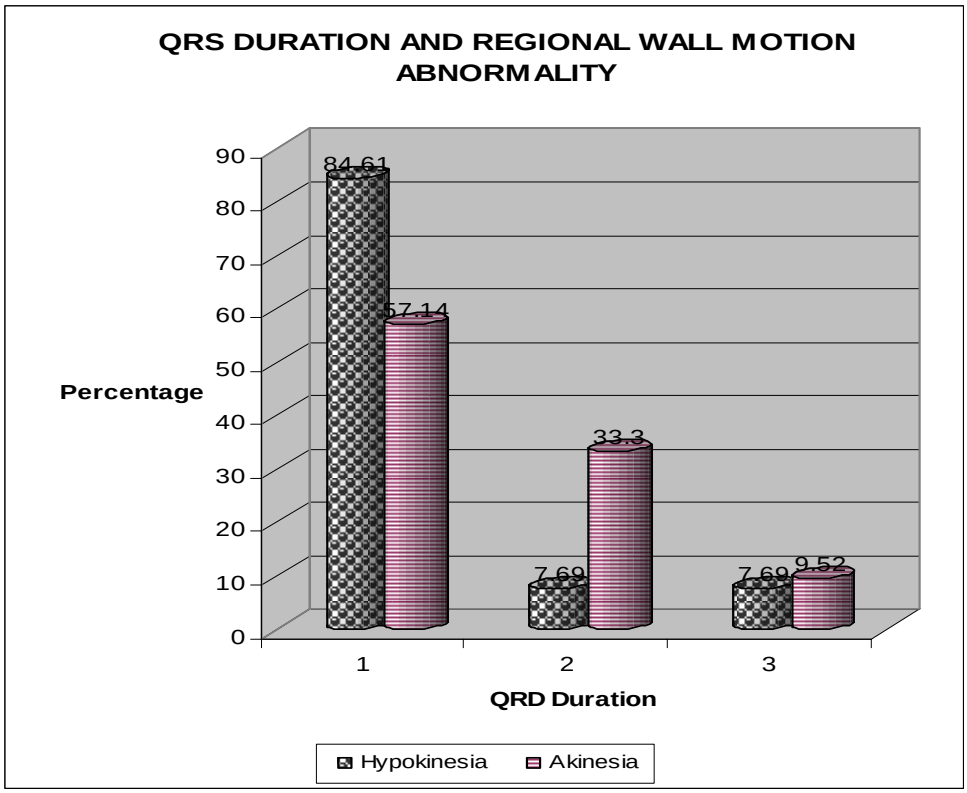
Ventricular ectopics were the most common arrhythmia observed. Maximum incidence of ventricular tachycardia was in group B (44.44%).

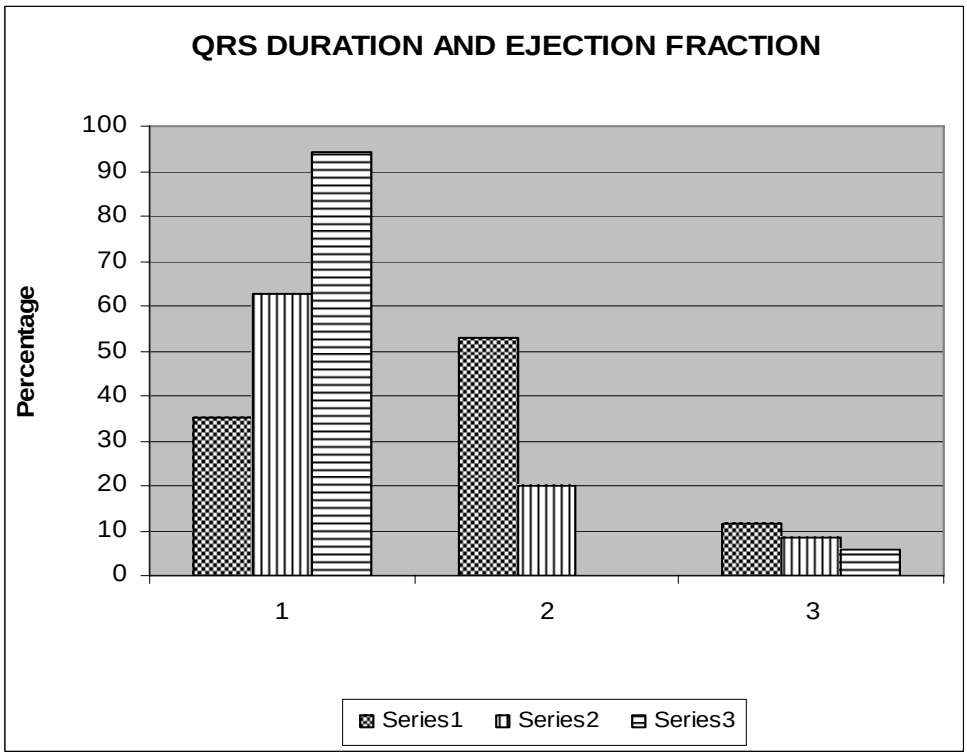
Table No. 6

QRS Duration and Regional Wall Motion Abnormality

RWMA	Group A (0.09) (n-49)		Group B (0.09 - 0.11) (n-18)		Group C (> 0.11) (11-8)		Total (n-69)	
	No.	%	No.	%	No.	%	No.	%
Hypokinesia	22	84.61	2	7.69	2	7.69	26	37.14
Akinesia	24	57.14	14	33.3	4	9.52	42	60.0
Normal	2	100.0	-	-	-	-	2	2.86
Total	48	68.57	16	22.85	6	8.5	70*	100.0

*5 Patients expired before Echocardiography


QRS DURATION AND REGIONAL WALL MOTION ABNORMALITY
Percentage
90
80
70
60
50
40
30
20
10
0
84.61
57.14
7.69
33.3
7.69
9.52
1
2
3
QRD Duration
Hypokinesia
Akinesia


QRS DURATION AND EJECTION FRACTION
Percentage
100
90
80
70
60
50
40
30
20
10
0
1
2
3
Series1
Series2
Series3

Akinesia was reported from 24 patients (50%) of group A, 14 patients (87.5%) of group B and 4 patients (66.67%) of group C. Hypokinesia was reported from 22 patients (46%) of group A, 2 patients (12.5%) of group B and 2 patients (33.33%) of group C. Two patients in Group A were having normal regional wall motion.

Table No. 7

QRS Duration and Ejection Fraction

Ejection Fraction (%)	Group A « 0.09) (n-49)		Group B (0.09 - 0.11) (n-18)		Group C (> 0.11) (n-8)		Total (n-70)	
	No.	%	No.	%	No.	%	No.	%
30-40 %	6	35.29	9	52.94	2	11.76	17	24.28
40-50 %	25	62.85	7	20.0	3	8.57	35	50.0
50-60 %	16	94.1	-	-	1	5.9	17	24.28
> 60%	1	100.0	-	-	-	-	1	1.42
Total	48	68.11	16	23.18	6	8.69	70*	100.0

*5 Patients expired before Echocardiography.

In Group A, patients with ejection fraction >50 was 35.45%. In Group C patients with ejection fraction >50 was 16.7% and in Group B patients had ejection fraction >50.

Table No. 8

Killip Class and Distribution of Cases in Myocardial Infarction in Relation to QRS Duration

Killip Class	A (n-49)		B (n-18)		C (n-8)			
	No.	%	No.	%	No.	%	No.	%
I	35	71.4	5	27.78	1	12.5	41	54.67
II	12	24.48	10	55.55	2	25.0	24	32.0
III	1	2.0	1	5.5	4	50.0	6	8.0
IV	1	2.0	2	11.11	1	12.5	4	5.3
Total	49	65.33	18	24.0	8	10.67	75	100.0

Chi square=39.21 p< 0.001 Highly Significant (Gr A vs B)
Chi square=104.88 p<0.001 Highly Significant (Gr A vs C)
Chi square=53.15 p<0.001 Highly Significant (Gr B vs C)

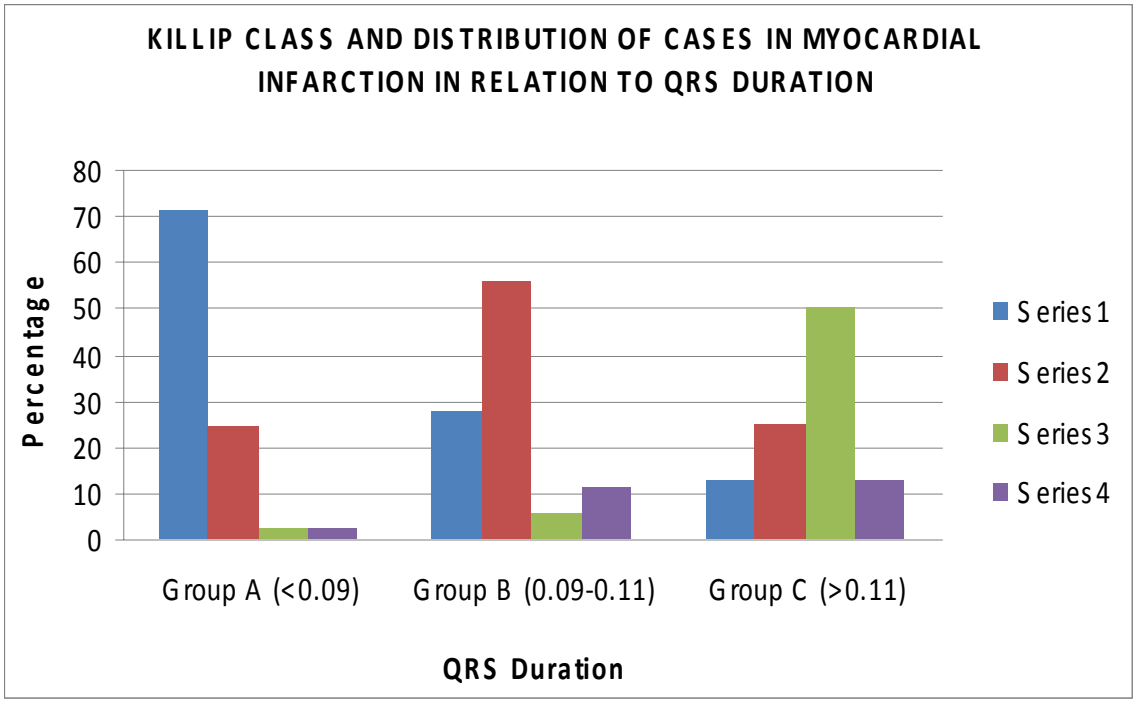
In group A 71.4 % of patients belonged to Killip class I as compared to 12.5% in group C. Patients with Killip class > II were 62.5% in group C, 16.6% in group B and 4% in group A.

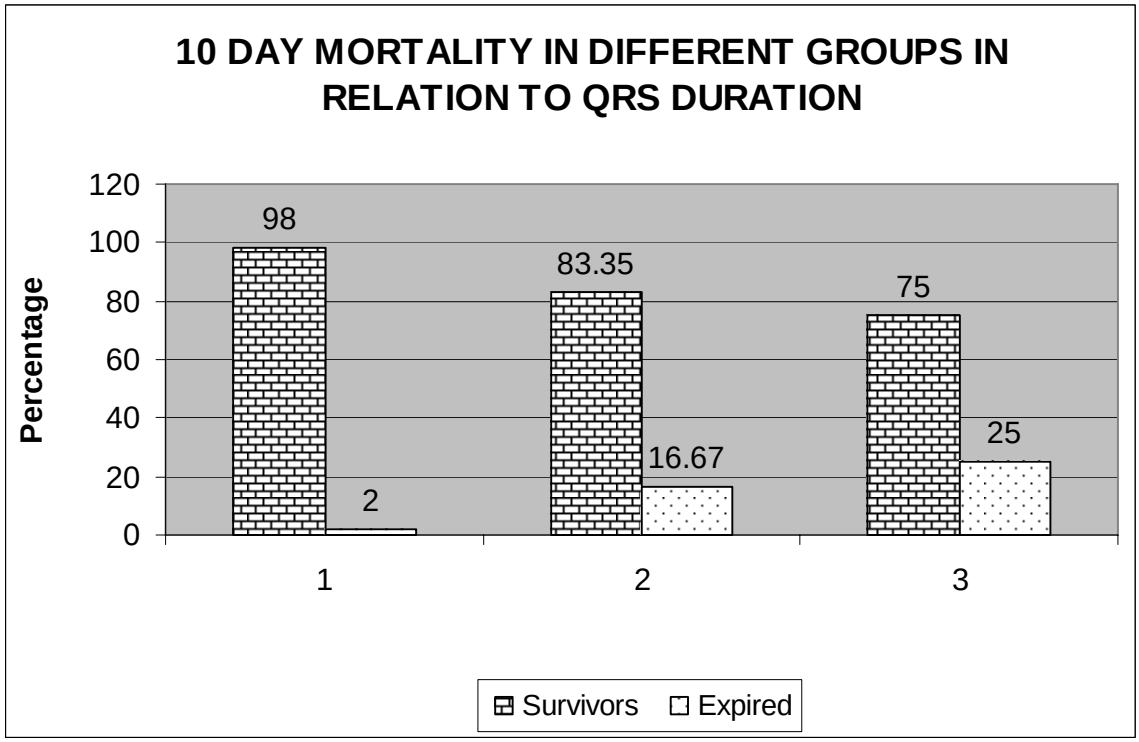
Table No. 9

Killip Class with Mean QRS Duration and Ejection Fraction in AWMI

Killip Class	Mean QRS Duration	Mean Ejection Fraction
I	0.08±0.01	47.60±9.82
II	0.09±0.01	42.20±4.78
III	0.09	43
N	0.10	Expired before Echo

r = 0.9- Significant


KILLIP CLASS AND DISTRIBUTION OF CASES IN MYOCARDIAL INFARCTION IN RELATION TO QRS DURATION
Percentage
80
70
60
50
40
30
20
10
0
Group A (<0.09)
Group B (0.09-0.11)
Group C (>0.11)
QRS Duration
Series 1
Series 2
Series 3
Series 4


10 DAY MORTALITY IN DIFFERENT GROUPS IN RELATION TO QRS DURATION
Percentage
120
100
80
60
40
20
0
98
2
83.35
16.67
75
25
1
2
3
Survivors
Expired

Mean QRS duration of class I patient was 0.08±0.01 as compared to 0.10 in Killip class IV patients. As QRS duration increases Killips Class also increases.

Table No. 10
10 Day Mortality in Different Groups in Relation to QRS Duration

QRS Duration	Survivors		Expired		Total
	No.	%	No.	%	
Group A (< 0.09)	48	98.0	I	2.0	49
Group B (0.09 - 0.11)	15	83.35	3	16.67	18
Group C(> 0.11)	6	75.0	2	25.0	8
Total	69	92.0	6	8.0	75

Chi square= 21.80 p< 0.001 Highly Significant

In the present study out of the 75 patients 6 (8.0%) expired; one from Group A (2.0%)/ 3 from group B (16.67%) and 2 (25.0%) from group C. In the mortality group, except one patient of AWMI, five of them expired before Echocardiography could be done and one after Echocardiography was done.

Table No. 11
Distribution of IWMI According to R/Q Ratio in Lead II

S.No.	R/Q Ratio in Lead II	No. of Cases	Percentage
1.	Group I (> 2)	22	46.80
2.	Group II (1-2)	21	44.68
3.	Group III (< 1)	4	8.51
Total		47	100.0

Maximum number of patients were in group I (46.80%) followed by group II (44.68%).

Table No. 12

R/Q Ratio and Complications at the Time of Admission

S. N.	Complications	R/Q Ratio		
		Group I (> 2) (n-22)	Group II (1-2) (n-21)	Group III (<1) (n-4)
1.	Hypotension	-	1 (4.76%)	1 (25.0%)
2.	Raised JVP	3 (13.63%)	4 (19.0%)	2 (50.0%)
3.	Arrhythmia	7 (31.81 %)	9 (42.8%)	2 (50.0%)

Chi square= 22.91 $p<0.001$ Highly Significant.

All patients of group III had different types of complications of myocardial infarction, 50% of them developed arrhythmia, 50% had raised JVP and 25% had hypotension as compared to group I where 31.8% patients had arrhythmia, 13.63% had

raised JVP and none of them had hypotension

Table No. 13
R/Q Ratio and Arrhythmia

Arrhythmia	Group I (R/Q > 2)		Group II (R/Q 1-2)		Group III (R/Q < 1)		Total (n-17)	
	No.	%	No.	%	No.	%	No.	%
S.V.E.	1	20.0	3	60.0	1	20.0	5	29.41
V.E.	4	57.14	3	42.85	-	-	7	41.17
S.V.T.	-	-	1	100.0	-	-	I	5.88
V.T.	1	33.3	1	33.3	1	33.3	3	17.64
C.H.B.	-	-	1	100.0	-	-	1	5.88

Most common arrhythmia reported was ventricular ectopics (41.1%) followed by supraventricular ectopics (29.4%). Out of 47 patients with inferior wall myocardial infarction only 3 patients (17.64%) had ventricular tachycardia, with 33.3% of patients in each group.

Table No. 14
R/Q Ratio and Ejection Fraction in Thrombolyzed and Non Thrombolyzed in Inferior Wal1 Myocardial Infarction

S. N.	IWMI (n-47)	*R/Q* Ratio (Mean±S.D.)	Ejection Fraction (Mean±S.D.)
1.	Thrombolyzed (n-26)	2.89±1.54	48.84±5.56
2.	Non-thrombolized (n-21)	1.89±1.0	46.89±6.78

t=2.57 p<0.05 Significant (Thrombolysed)
t=4.19 p<0.001 Highly Significant (Non Thrombolysed)

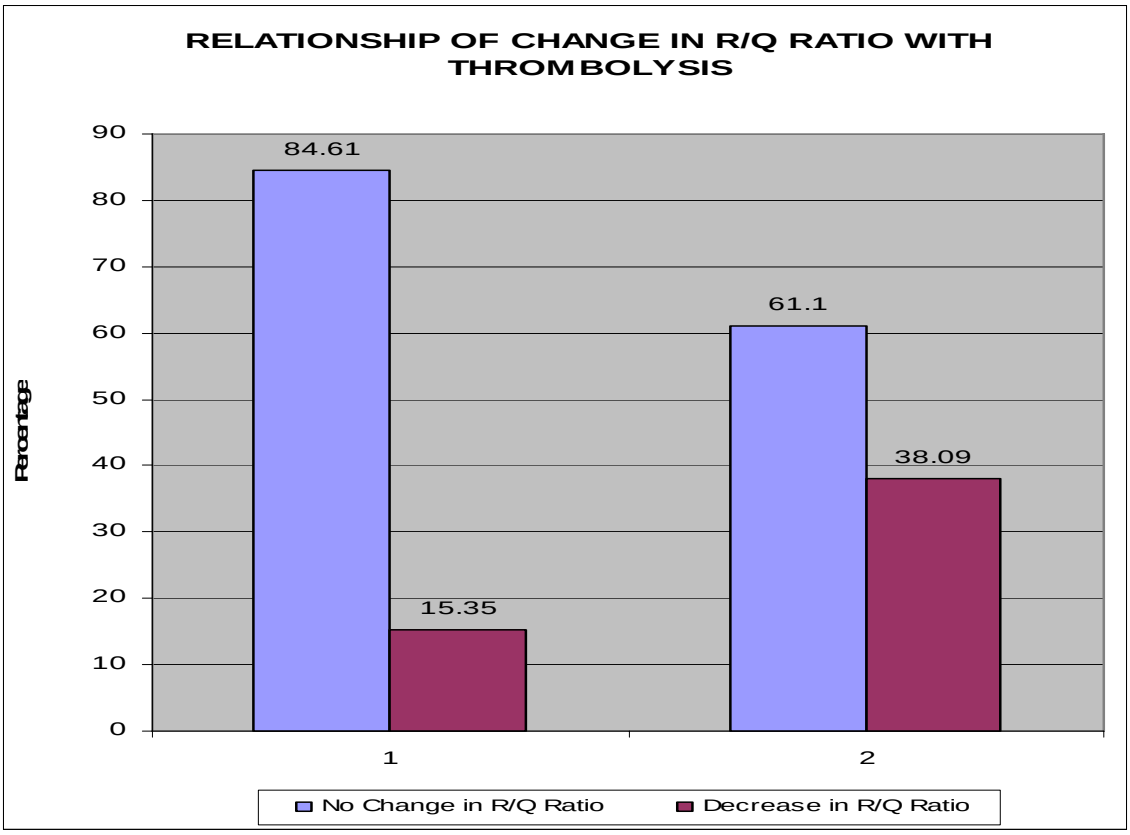
There is statistically significant difference in mean R/Q ratio and EF in thrombolysed and non thrombolysed patients of inferior wall myocardial infarction.

Table No. 15
Relationship of Change in R/Q Ratio with Thrombolysis

Changes in R/Q Ratio from 3rd to 7th Day	Thrombolyzed (n-26)		Non-thrombolyzed (n-21)	
	No.	%	No.	%
Increase in R/ Q Ratio	-	-	-	-
No Change in R/Q Ratio	22	84.61	13	61.10
Decrease in R/Q Ratio	4	15.35	8	38.09

Chi square=9.92 $p<0.05$ Significant

Out of 26 patients of inferior wall myocardial infarction thrombolyzed 22 patients (84.61 %) had no change in R/Q ratio from 3rd day to 7th day. But 4 patients (15.35%) showed a decrease in R/Q ratio on 7th day as compared to R/Q ratio on 3rd day. In non-thrombolyzed patients of IWMI 8 patients (38.09%) showed a decrease in R/Q ratio on 7th day as compared to R/Q ratio on 3rd day. So significantly less number of patients showed decrease in R/Q ratio in thrombolyzed as compared to non-thrombolyzed patients.


RELATIONSHIP OF CHANGE IN R/Q RATIO WITH THROMBOLYSIS
Percentage
90
80
70
60
50
40
30
20
10
0
84.61
15.35
61.1
38.09
1
2
No Change in R/Q Ratio
Decrease in R/Q Ratio

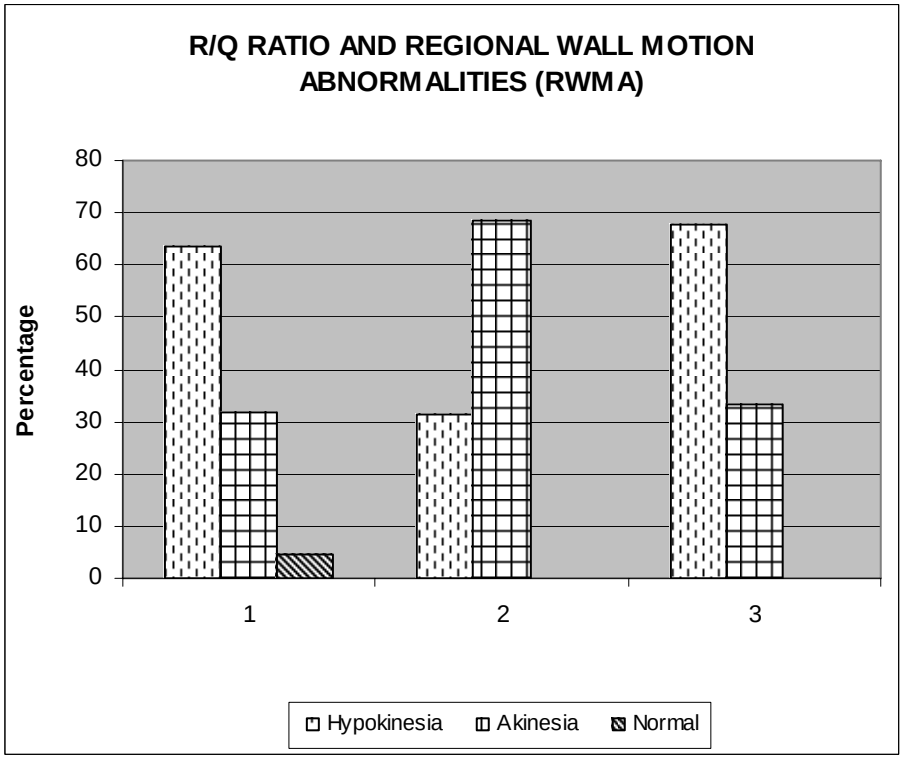

R/Q RATIO AND REGIONAL WALL MOTION ABNORMALITIES (RWMA)
Percentage
80
70
60
50
40
30
20
10
0
1
2
3
Hypokinesia
Akinesia
Normal

Table No. 16
R/Q Ratio and Regional Wall Motion Abnormalities (RWMA)

RWMA	Group I (>2)		Group II (1-2)		Group III «1)	
	No.	%	No.	%	No.	%
Hypokinesia (n-22)	14	63.64	6	31.57	2	66.67
Akinesia (n-21)	7	31.8	13	68.43	1	33.3
Normal (n-1)	1	4.54	-	-	-	-
Total (n-44)	22	100.0	19	100.0	3	100.0

*3 patients of IWMI expired befor Echocardiography.

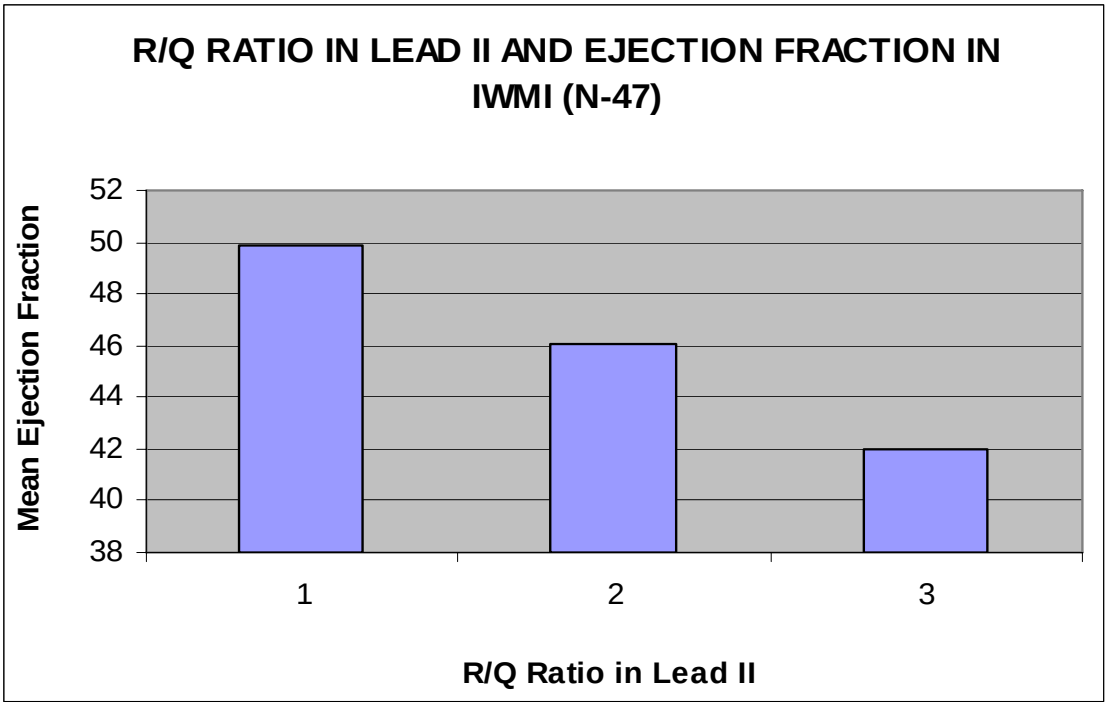
Chi square=10.23 p<0.05 Significant (I vs III)
Chi square=12.99 p<0.05 Significant (II vs III)
Chi square=25.69 p<0.001 Highly Significant (I vs II)

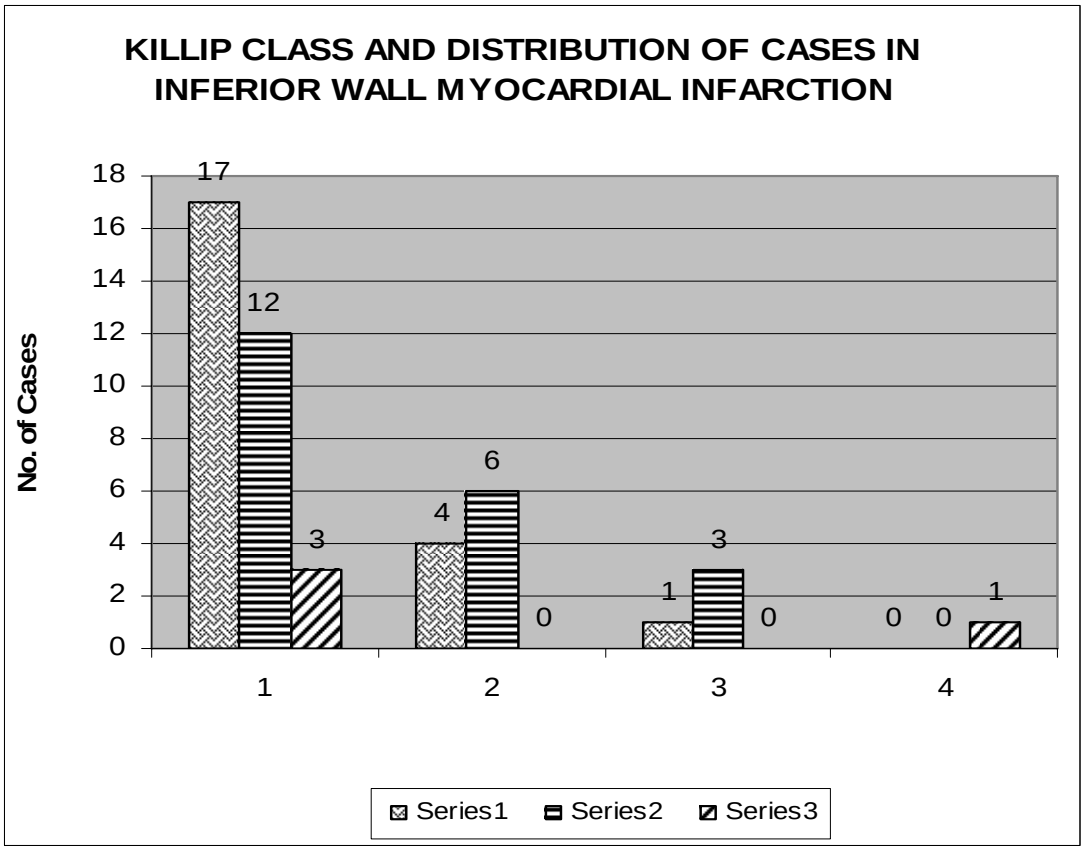
Out of 22 patients in group I, 14patients (63.64%) had hypokinesia and 7 patients (31.8%) had akinesia of inferior segment of left ventricle. Out of 44 patients of IWMI; 22 patients had hypokinesia in which 14 patients (63.64 %) belonging to group I; only 2 patients (9.0%) belonged to group III.

Table No. 17
R/Q Ratio in Lead II and Ejection Fraction in IWMI

S. No.	RlQ Ration in Lead II	No. of Cases	Mean Ejection Fraction
1.	Group I (> 2)	22	49.91±5.99
2.	Group II (1-2)	21	46.10±5.84
3.	Group III (< 1)	4	42±6.32

t=2.41 p<0.05 Significant (I vs III)
t=2.11 p<0.05 Significant (I vs II)
t=1.27 p>0.05 Insignificant (II vs III)


R/Q RATIO IN LEAD II AND EJECTION FRACTION IN IWMI (N-47)
Mean Ejection Fraction
52
50
48
46
44
42
40
38
1
2
3
R/Q Ratio in Lead II


KILLIP CLASS AND DISTRIBUTION OF CASES IN INFERIOR WALL MYOCARDIAL INFARCTION
No. of Cases
18
16
14
12
10
8
6
4
2
0
17
12
3
4
6
0
1
3
0
0
0
1
1
2
3
4
Series1
Series2
Series3

Among the various groups, maximum mean ejection fraction was reported in group I i.e. R/Q > 2. Lowest mean ejection fraction was in patients with R/Q ration < 1 (group III).

Table No. 18
Killip Class and Distribution of Cases in IWMI

Killip Class	Group I (R/Q> 2)		Group II (R/Q 1-2)		Group III (R/Q < 1)	
	No.	%	No.	%	No.	%
I (n=32)	17	77.2	12	57.0	3	75.0
II (n=10)	4	18.18	6	28.5	-	-
III (n=4)	1	4.54	3	14.28	-	-
IV (n=1)	-	-	-	-	1	25.0
Total (n=47)	22	100.0	21	100.0	4	100.0

Chi square=70.23 p<0.001 Highly Significant (Gr I vs Gr III)
Chi square=10.36 p<0.05 Significant(Gr I vs Gr II)

Out of 32 cases of Killip class 117 (53.12%) patients from Group I and 3 (9.37%) patients are from group III. Only one case of Killip class IV was reported from the present study, which was from group III. Majority of group II patients were in Killip class I and II (85.5%).

Table No. 19
Killip Class with Mean R/Q Ratio and Ejection Fraction in IWMI

Killip Class	Mean R/Q Ratio	Mean Ejection Fraction
I	2.66 ±1.49	49.55 ±5.22
II	2.19 ±1.02	46.64 ±6.61
III	2.0 ±1.73	40.0 ±5.29
IV	0	38

r =-1.0, insignificant r = -1.0, insignificant

Mean R/Q ratio of Killip class I was 2.66 ±1.49 against 2.0 ±l.73 in Killip class III. Mean ejection fraction of Killip class I was 49.55 ±5.22 and 40.0 ±5.29 in Killip class III. There was a weak correlation between mean R/Q ratio and mean ejection fraction with Killip class.

Table No. 20
Comparison of R/Q Ratio and QRS Duration in Relationship to Ejection Fraction

S.No.	R/Q Ratio	Mean Ejection Fraction	QRS Duration	Mean Ejection Fraction
1.	Group I (> 2)	49.91 ±5.99	Group A (< 0.09)	48.96 ±6.53
2.	Group II (1-2)	46.10 ±5.84	Group B (0.09 - 0.11)	40.94 ±3.90
3.	Group III (<1)	42 ±6.32	Group C (> 0.11)	43.17 ±4.83

Mean ejection fraction of group I was 49.91 ±5.99 and group A was 48.86 ±6.53. Mean ejection fraction of group III was 42 ±6.32 and in group C it was 43.17 ±4.83, as *R/Q* decreases the reduction in mean ejection fraction was also significant. Similar to this prolongation of QRS duration also correlates with decrease in mean ejection fraction. Intermediate QRS duration prolongation (0.09-

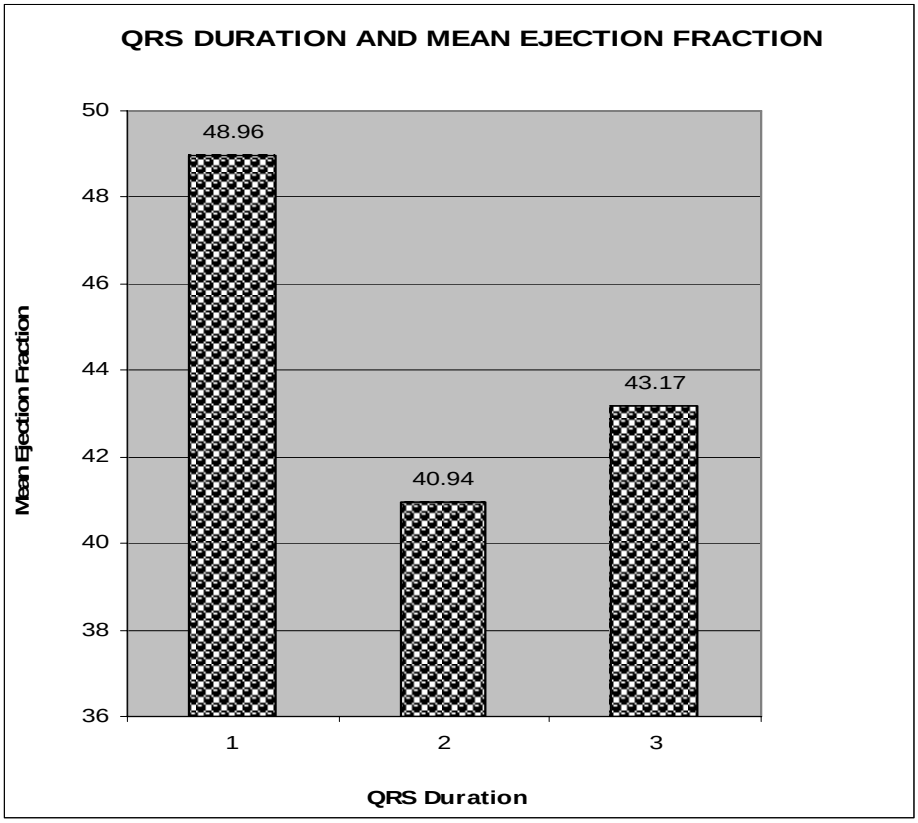
0.11) in Group B had lowest mean ejection fraction.

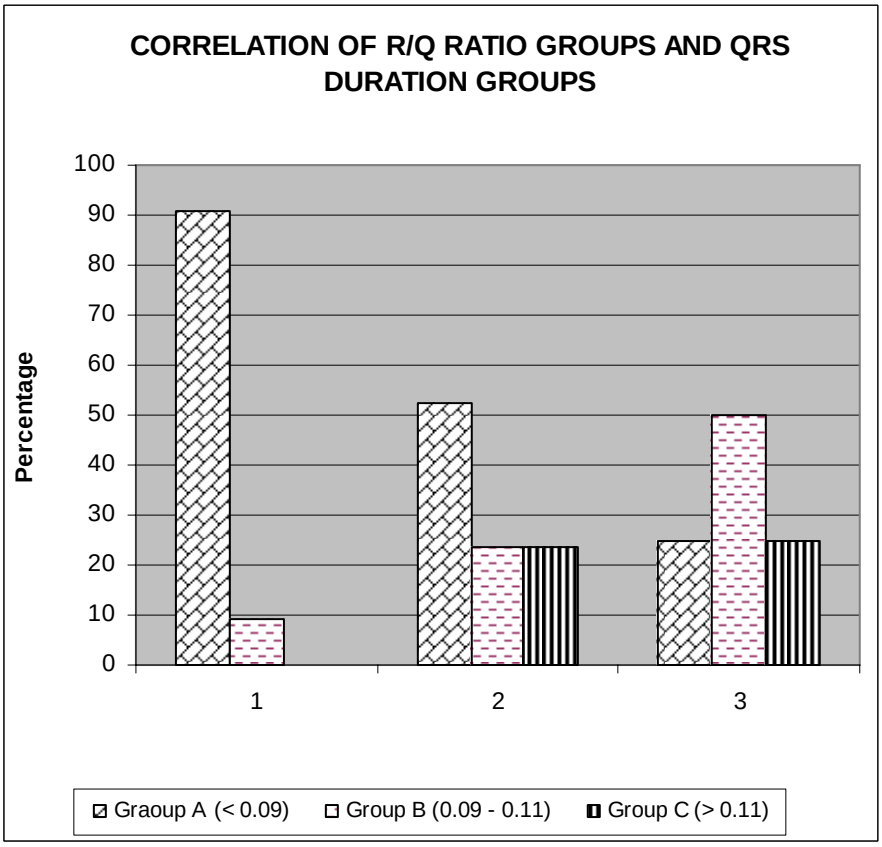
Table No. 21
Correlation of R/Q Ratio Groups with QRS Duration Groups in IWMI

R/Q Ratio	No .of Patients	QRS Duration	No. of Patients	%
Group I (R/Q> 2)	22	Group A «0.09)	20	90.90
		Group B (0.09-0.11)	2	9.10
		Group C (>0.11)	0	0
Group II (R/Q 1- 2)	21	Group A (>0.09)	11	52.4
		Group B (0.09-0.11)	5	23.80
		Group C (>0.11)	5	23.80
Group III (R/Q < 1)	4	Group A (<0.09)	1	25.0
		Group B (0.09-0.11)	2	50.0
		Group C (>0.11)	1	25.0

Percentage of patients with QRS duration < 0.09 sec was 90.90% in group I, 52.40% in group II and 25% in group III.

In group III (R/Q<l) 25% patients belong to group A and 25% belong to group C and none of patients in group I had QRS duration> 0.11 sec.


QRS DURATION AND MEAN EJECTION FRACTION
Mean Ejection Fraction
50
48
46
44
42
40
38
36
48.96
40.94
43.17
1
2
3
QRS Duration


CORRELATION OF R/Q RATIO GROUPS AND QRS DURATION GROUPS
Percentage
100
90
80
70
60
50
40
30
20
10
0
1
2
3
Graoup A (< 0.09)
Group B (0.09 - 0.11)
Group C (> 0.11)

DISCUSSION

The present series “Study of QRS Duration and R/Q Ratio in the Assessment of Severity of Acute Myocardial Infarction” was carried out on 75 patients admitted in ICCU of Department of Medicine, Government Royapettah Hospital, Kilpauk Medical College, Chennai-10.

For the study of “QRS Duration” in the severity of myocardial infarction (both AWMI and IWMI) patients were divided into 3 groups.

Group A With QRS duration < 0.09 Sec.

Group B With QRS duration 0.09 - 0.11 see (intermediate QRS prolongation)

Group C With QRS duration > 0.11 see (Significant QRS prolongation)

For the study; patients of inferior wan myocardial infarction were divided into 3 groups according to “R/Q ratio” in lead II on 3rd day.

Group I Includes patients of IWMI with R/Q ratio> 2

Group II Includes patients of IWMI with R/Q ratio 1- 2

Group III Includes patients of IWMI with R/Q ratio < 1

TYPE OF MYOCARDIAL INFARCTION

In the present study 44 patients (58.67%) were having inferior wall myocardial infarction 28 patients (37.33%) were having anterior wall myocardial infarction and 3 (4.0%) were having both anterior and inferior wall myocardial infarction.

DISTRIBUTION OF EJECTION FRACTION

In the present study 34.61% of anterior wall myocardial infarction patients were having LVEF of < 40% as compared to 17.07% of patients of inferior wall myocardial infarction. The percentage of patients of anterior wall myocardial infarction with ejection fraction of > 50% was only 15.38% as compared to 31.07% in inferior wall myocardial infarction. So statistically significant lower ejection fraction in anterior wall myocardial infarction as compared to IWMI ($p< 0.001$) was seen.

The observation was similar to that of Mark et al (1987) (53) who observed that there is greater left ventricular involvement and dysfunction in patients with anterior myocardial infarction of equivalent enzymatic size.

Eaton et al (1979) (54) in a limited series of 28 patients showed that anterior infarcts are more at risk of expanding with thinning of the infarct zone combined with acute regional dilatation than inferior infarcts. It is anticipated that this process would lead to greater left ventricular regional wall motion abnormality both at rest and during exercise.

Strass et al (1980) (55) observed that there was greater involvement and reduced function of the left ventricle in patients with anterior infarction compared to those with inferior infarction of equivalent enzymatic size.

QRS DURATION AND DISTRIBUTION OF MYOCARDIAL INFARCTION:

In the present study most of the patients of inferior as well as anterior wall myocardial infarction were distributed in group A (QRS < 0.09 sec.) 40% of AWMI patients were having QRS duration > 0.09 seconds as compared to 30% of patients in IWMI ($p<0.05$).

QRS DURATION AND ARRHYTHMIA IN MYOCARDIAL INFARCTION:

Ventricular ectopics were the most common arrhythmia observed in relationship to QRS duration groups. Maximum incidence of ventricular tachycardia (44.44%) was in group B (QRS 0.09-0.11 sec.). Out of the 8 patients in group C (QRS > 0.11 sec) 2 patients were (25.0%) having ventricular tachycardia as com pared to 3 patients (6.38%) in group A (< 0.09 sec.) so statistically significant high incidence of malignant arrhythmias was noted in patients of intermediate QRS prolongation (0.09 - 0.11 sec) and significant QRS prolongation (> 0.11 see) in the present study.

Previous study by Pudil et al (2001) (38) showed that in hospital complication including asystole, ventricular tachycardia, ventricular fibrillation, congestive heart failure were more common among patients with prolonged QRS duration.

Prolonged QRS duration was a significant predictor of sustained monomorphic VT inducibility (p<0.00l) (Relative risk 3.290, 95% confidence interval 2.185 to 4.953 for prolonged vs normal QRS duration) (44).

At multivariate analysis, only low left ventricular ejection fraction, prolonged QRS duration, reduced heart rate variability index, and detection of approximately 2 runs of unsustained VT per monitoring had an independent relation to late arrhythmic events (45).

REGIONAL WALL MOTION ABNORMALITY AND EJECTION FRACTION IN RELATION TO QRS DURATION:

In the present study akinesia was reported from 14 patients (87.5%) of group B (0.09 - 0.11 sec.), 4 patients (66.67%) of group C (> 0.11 see) and 24 patients (50%) of group A « 0.09 see); hypokinesia was reported from 2 patients (12.5%) of group B, 2 patients of group C (33.33%) and 22 patients (46.0%) of group A. Hence, regional wall motion abnormality was having a significant relationship with QRS duration. Increased QRS duration had an increased incidence of akinesia in the present study (p< 0.001).

In the present study patients with ejection fraction of >50% in group A was 35.42% in group C 16.67%. None of the patients in Group B was having ejection fraction of> 50%.

In a previous study by Brilakis et al (46) QRS duration> 100 msec on standard 12 lead ECG was a specific indicator of left ventricular dysfunction (EF< 45%).

In 27 out of 28 postmyocardial patients in whom left ventricular systolic dysfunction (defined as ejection fraction of < 40%) developed; QRS duration increased from 107±12 milliseconds to128±18 milliseconds.

Pudil et al (2001) (38) showed that a QRS of <0.09 sec on admission electrocardiogram is indicative of a relatively benign outcome compared with a QRS of >0.09 sec. Patients with intermediate QRS prolongation (0.09 - 0.11 sec.) also associated with decreased ejection fraction and increased incidence of akinesia. Hence intermediate QRS duration prolongation had also statistically significant relationship with ejection fraction and RWMA in a similar way to significant QRS prolongation (> 0.11 sec.).

QRS DURATION AND KILLIP CLASS:

In the present study in group A (< 0.09 sec.) 71.4% (n=35) of patients were belonging to Killip class I as compared to 12.5% in group C. Patients with Killip class> II were 4% in group A, 62.5% in group C and 16.6% in group B. These results are statistically significant (p< 0.05).

In the present study mean QRS duration of class I patient was 0.08±0.01 as compared to 0.10 in Killip class IV. As Killip class of patient increases mean QRS duration also increases.

Above observations go in agreement with previous study by Brilakis et at (2002) (46) in which patients with QRS duration < 100 milliseconds to be in Killip class II, III or IV at presentation. Patients with QRS duration < 100 milliseconds, percentage of patients with Killip class> II was 16.7% against 32% in patients with QRS duration > 100 millisec.

Michaelides et al (1993) (42) reported that exercise induced QRS prolongation was proportional to the number of coronary arteries with > 70% stenosis. Mean QRS prolongation was 4.8±7.5 milliseconds in patients with I-vessel disease, 7.8±11.8 milliseconds in patients with 2-vessel disease and 13.3±12.1 milliseconds in patients with 3-vessel disease. Study by Brilakis. et al (2002) (46) showed increased QRS duration was strongly associated with heart failure as evidenced by worse Killip class.

MORTALITY IN RELATION TO QRS DURATION:

In the present study, out of the 75 patients 6 (8.0%) expired. One from group A (2.0%), 3 from group B (16.67%), and 2 (25.0%) from group C. In the mortality group, except one patient of AWMI rest of them expired before echocardiography could be done. So correlation with ejection fraction and R/Q ratio was not possible; hence mortality was analysed on the basis of QR5 duration. Analysis of present study data shows that increased QRS duration had a significant statistical relationship with mortality ($p< 0.05$).

QRS duration measured on a standard ECG remains a powerful predictor of mortality after adjustment for ejection fraction and other clinical covariates. Data in a large population of post infarction patients indicates that QRS of 0.12 seconds is associated with hazard ratio of 1.7; $p = 0.001$. Above observations in the present study i.e; highest mortality 25% in group C patients (QRS > 0.11 sec.) is in agreement with the observations of Fadl et al (2003). (47)

QRS duration remains a very powerful predictor of future cardiac events in post infarction patients. QRS duration reflects well the magnitude of left ventricular

dysfunction and therefore not surprisingly is a powerful predictor of mortality in post infarction patients. (40)

In a previous study by Pudil et al (2001) (38) 7 day mortality of patients with QRS duration (<0.09 sec) was < l%, 6% in 0.09-0.011 see group and 18% in > 0.11 see group. So the present study observations go in agreement with the study of Pudil et al (2001) (38).

R/Q RATIO AND COMPLICATIONS OF MYOCARDIAL INFARCTION

In the present study statistically significant relation between R/Q ratio and complications of myocardial infarction was present. All patients of group III (R/Q < 1) were having different types of complications of myocardial infarction, 50% of them developed arrhythmia, 50% having raised JVP, and 25% having hypotension as compared to group I (> 2), where 31.8% of patients where having arrhythmia, 13.63 % having raised JVP and none of them had hypotension.

In a previous study by Alexander Arditti et al (1985) (50) no patient with R/Q ratio> 2 developed signs of heart failure, compared to 2 patients in group II (R/Q ratio 1-2) and 4 patients in group In (R/Q ratio < 1).

In another study by Eliezer Klainman et al (1988)(51) showed that clear tendency for more incidence of complications of myocardial infarction in group III (R/Q ratio < 1) as compared to group I (R/Q ratio> 2).

In the present study the most common arrhythmia reported was ventricular ectopics (41.1%) followed by supraventricular ectopics (29.41 %).

Out of the 47 patients of inferior wall myocardial infarction only 3 patients (6.38%) were having ventricular tachycardia with equal percentage of patients in each group.

These findings go in agreement with Alexander Arditti et al (1985)(50) in that there were no statistical differences between three groups of inferior wall myocardial infarction in the incidence of ventricular tachycardia and other arrhythmias. Ventricular tachycardia was reported in 5 patients out of 39 in R/Q > 2 group and 5 patients out of 29 in R/Q < 1 group.

Data from Gusto I trial indicates that in thrombolytic era sustained VT occurs in 3.5% of patients, ventricular fibrillation in 4.1% of patients and a combination of sustained ventricular tachycardia and ventricular fibrillation in 2.7% of patients.

Statistically significant relation between R/Q ratio and mean ejection fraction was seen in the present study. Mean ejection fraction of patients with R/Q ratio> 2 (group I) was 49.91±5.99, 46.10±5.84 in patients with R/Q ratio 1-2 (group II) and 42 ±6.32 in patients of R/Q ratio < 1 (group III).

In the previous study by Alexander Arditti et al (1985) (50) mean ejection fraction in group III (R/Q ratio<1) was 51.50±9.40 as compared to mean ejection fraction 60.15±8.90 in group I (R/Q ratio>2). Intermediate group was having mean ejection fraction of 51.20±16.50.

In 1974 Miller et al (56) demonstrated that, the presence of a pathologic Q wave was a specific indicator of dyssynergy and that the sensitivity of a QRS complex for predicting wall motion abnormalities increased with severity of dyssenergy. ldekar et al

(1978)(57) examined the histopathological findings in 24 patients with ischemic heart disease and dyssynergy demonstrated by contrast ventriculography. They noted that the degree of dyssynergy was directly related to the percentage of fibrosis and the percentage of the total left ventricle occupied by fibrotic tissue was significantly related to Global left ventricular ejection.

R/Q RATIO AND REGIONAL WALL MOTION ABNORMALITIES:

In the present study, out of 22 patients in group I (R/Q ratio>2), 14 patients (63.64%) were having hypokinesia and 7 patients (31.8%) were having akinesia of inferior segment of left ventricle. Out of the 44 patients of inferior wall myocardial infarction 22 patients were having hypokinesia in which 14 patients (63.64%) belonging to group I (R/Q ratio> 2) only 2 patients (9.0%) belonged to group III (R/Q ratio < 1). So present study shows that with good R/Q ratio i.e. > 2 severity of regional wall motion abnormality decreases. As R/Q ratio decreases, severity of regional wall motion abnormality increases.

This finding is in agreement with the previous study of Alexander Arditti et al (1985) (50) which showed that group I (R/Q ratio. > 2) was notable only for the presence of hypokinesia of inferior wall; while other segments are minimally affected. Group II (R/Q ratio 1-2) showed a higher incidence of akinesia, group III (R/Q ratio < 1) showed higher incidence of akinesia of different segments of inferior wall of left ventricle.

In Inferior wall myocardial infarction a QS complex is usually present in lead III and aVF, standard lead II usually reflects Qr and QR complex. In inferior wall

myocardial infarction, among the inferior leads tallest terminal ’R’ wave is seen in lead II.

We can explain this distribution of terminal ‘R” wave by considering the anatomy of inferior wall. We can divide the inferior wall into right lateral and left lateral region. Lead III is oriented to right lateral region and lead II to left lateral region. The brunt of infarction affects the right lateral region of inferior wall with sparing of left lateral region. So lead III reflects the largest and widest QS complex. Lead II however is oriented to left lateral region of inferior wall reflects the potentials of healthy overlying muscle as a terminal ‘R’ wave. So loss of ‘R’ wave in lead II occurs mainly in extensive inferior wall myocardial infarction. So ampliitude of ‘Q’ and ‘R’ wave in lead II is a measure of severity of inferior wall myocardial infarction (48).

KILLIP CLASS AND R/Q RATIO

In present study, distribution of inferior wall myocardial infarction in relationship to Killip class was statistically significant. Out of 32 cases of Killip class I, 17 (53.12%) are from group I (R/Q ratio> 2) and 3 cases (9.37%) are from group III (R/Q ratio < 1). Only one case of Killip class IV was reported from the present study, which was from group III (R/Q ratio < 1). Majority of group II (R/Q ratio 1-2) patients are in Killip class I and II (85.5%). ($p < 0.05$).

In the previous study by Eliezer Klainman et al (1988) (51) showed that on decrease of R/Q ratio in lead II, Killip class was on rising trend. Study by Alexander Arditti et al (1985) (50) showed that in a group of inferior wall myocardial infarction with R/Q ratio>2, majority of the patients were in Killip class I, none developed

congestive heart failure. In patients with R/Q ratio 1-2 majority of patients are in Killip class I and II. Patients with R/Q ratio < 1, majority of patients were in Killip class II and III.

RATIO AND THROMBOLYSIS IN IWMI:

Present study shows that there is a statistically significant difference in R/Q ratio in thrombolyzed and non thrombolyzed patients of inferior wall myocardial infarction ($p< 0.05$).

Out of 26 patients of inferior wall myocardial infarction thrombolyzed 22 patients (84.61%) had no change in R/Q ratio on 7th day compared to 3rd day, 4 patients (15.35%) showed a lower R/Q ratio on 7th day as compared to 3rd day of hospitalisation.

But in non-thrombolyzed cases of inferior wall myocardial infarction 8 patients (38.09%) had a lower R/Q ratio on 7th day as compared to R/Q ratio on 3rd day. So these results show that lower R/Q ratio on 7th day in non-thrombolyzd patients as compared to thrombolyzed patients of inferior wall myocardial infarction was statistically significant ($p< 0.05$).

We can explain this observation in that, lead II is oriented to left lateral region of inferior wall and lead III to right lateral lesion. If proper thrombolysis was done, the brunt of infarction restricted to the right lateral region of inferior wall. SO R/Q ratio not decreases with time because thrombolysis prevent the extend of infarction to left lateral region and reflects the potentials of healthy overlying muscle as a terminal ‘R’ wave but in non thrombolysed patients extend of infarction occurs with loss of ‘R’ wave and development of ‘Q’ wave.

MEAN EJECTION FRACTION IN RELATION TO R/Q AND QRS DURATION:

In the present study mean ejection fraction of group I (R/Q ratio> 2) was 49.91±5.99 and group A (QRS duration < 0.09 seconds) was 48.86±6.53. Mean ejection fraction of group III (R/Q ratio < 1) was 42±6.32 and in group C (QRS duration> 0.11sec) it was 43.17±4.83. As R/Q decreases; statistically significant reduction of mean ejection fraction also occurs. Similar to this, prolongation of QRS duration also correlates with decrease in mean ejection fraction. Intermediate QRS prolongation (0.09 - 0.11secs) i.e. group B were having lowest mean ejection fraction.

In previous study by Alexander Arditti et al (1985) (50) mean ejection fraction was 60.15±8.90 in patients with R/Q ratio>2 and as R/Q decreases to < 1 mean ejection fraction decreased to 51.50±9.40. Horwich et al, (2003) (44) showed that patients with prolonged QRS duration had lower left ventricular ejection fraction.

Study by Pudil et al (2001) (38) showed that similar to significant QRS duration prolongation (> 0.11 sec.); intermediate QRS duration prolongation (0.09 - 0.011 sec.) on surface electrocardiograms was associated with left ventricular dysfunction. Murkofsky et al (1998) (40) showed that QRS duration> 0.1 sec was associated with decreased left ventricular ejection fraction.

DISTRIBUTION OF QRS DURATION GROUPS IN R/Q RATIO GROUPS IN INFERIOR WALL MYOCARDIAL INFARCTION:

Out of 22 patients of group I (R/Q ratio> 2) 90% of patients are in group A (QRS < 0.09 sec.), none of them had QRS duration > 0.11 sec (Group C). But in group III (R/Q

ratio < 1) 25% patients are of group A (< 0.09 sec.) and 25% are of group C (> 0.11 sec.). These results shows that as R/Q ratio decreases from> 2 in group I to < 1 in group IIII percentage of distribution of patients with QRS prolongation increases (> 0.09 see.) and patients with normal QRS duration (<0.09 sec) decreases. So the above results are statistically highly significant (p< 0.001). Analysis of present study shows that both lower R/Q ratio in lead II and prolonged QRS duration signifies the extent of inferior myocardial infarction. So estimation of R/Q ratio in lead II or QRS duration; in standard 12 lead ECG serves as an extremely useful tool in the risk stratification after inferior myocardial infarction.

SUMMARY AND CONCLUSION

The present series “Study of QRS Duration and R/Q Ratio in the Assessment of Severity of Acute Myocardial Infarction” was carried out on 75 patients admitted in the ICCU of Department of Medicine, Government Royapettah Hospital, Kilpauk Medical College, Chennai -10.

For the study of “QRS Duration” in the severity of myocardial infarction (both AWMI and IWMI) patients were divided into 3 groups.

Group A With QRS duration < 0.09 Sec.

Group B With QRS duration 0.09 - 0.11 sec (intermediate QRS prolongation)

Group C With QRS duration > 0.11 sec (Significant QRS prolongation)

For the study; patients of inferior wan myocardial infarction were divided into groups according to “R/Q ratio” in lead II on 3rd day.

Group I Includes patients of IWMI with R/Q ratio> 2

Group II Includes patients of IWMI with R/Q ratio 1- 2

Group III Includes patients of IWMI with R/Q ratio < 1

The patients were selected after application of the following exclusion criteria - patients with ECGs, showing evidence of LBBB, RBBB, LAHB, LVH, old myocardial infarction, pacemaker dependence.

(1) In the present study 34.61% of anterior wall myocardial infarction were having LVEF of < 40% as compared to 17.07% of patients of inferior wall myocardial infarction. The percentage of patients of anterior wall myocardial infarction with ejection fraction of> 50% was only 15.38% as compared to 31.07% in inferior wall myocardial infarction so statistically significant lower ejection fraction in anterior wall myocardial infarction as compared to IWMI.

(2) Ventricular ectopics were the most common arrhythmia observed in relationship to QRS duration groups. Maximum incidence of ventricular tachycardia (44.44 %) was in group B (QRS 0.09-0.11 sec.). Out of the 8 patients in group C (QRS > 0.11 sec.) 2 patients were (25.0%) having ventricular tachycardia as compared to 3 patients (6.38%) in group A (<0.09 sec) so statistically significant high incidence of malignant arrhythmias in patients of intermediate QRS prolongation (0.09 - 0.11 see) and significant QRS prolongation (> 0.11 see) was noted in the present study.

(3) Akinesia was reported from 14 patients (87.5%) of group B (0.09 - 0.11 sec.), 4 patients (66.67%) of group C (> 0.11 see) and 24 patients (50%) of group A « 0.09 see); hypokinesia was reported from 2 patients (12.5%) of group B, 2 patients of group C (33.33%) and 22 patients (46.0%) of group A. Hence, regional wall motion abnormality had a statistically significant relationship with QRS duration. Increased QRS duration had an increased incidence of akinesia in the present study (p< 0.001).

(4) Statistically significant relationship was present between ejection fraction and QRS duration in the present study Patients with ejection fraction of> 50% in group A was 35.42% and in group C, it was 16.67%.

(5) In the present study in group A 71.4 % of patients were belonging to Killip class I as compared to 12.5% in group C. Patients with Killip class> II were 4% in group A 62.5% in group C and 16.6% in group B.

(6) Mean QRS duration of class I patients was 0.08±0.01 as compared to 0.10 in Killip class IV. As Killip class of patient increases mean QRS duration also increases. So the present study demonstrated that there was a statistically significant relation between QRS duration and Killip class of myocardial infarction.

(7) Mean ejection fraction of group A (<0.09 seconds) patients was 48.86±6.53, 40.94±3.90 in group B and 43.17±4.83 in group C, so both intermediate and significant QRS prolongation was having decreased mean ejection fraction so intermediate QRS prolongation is as significant as that of significant QRS prolongation in assessing LV function.

(8) In the present study statistically significant relation between R/Q ratio and complications of myocardial infarction was present. All patients of group III (R/Q < 1) were having different types of complications of myocardial infarction, 50% of them developed arrhythmia, 50% having raised JVP, and 25% having hypotension as compared to group I (> 2), where 31.8% of patients where having arrhythmia, 13.63% having raised JVP and none of them had hypotension.

(9) In the present study in inferior wall myocardial infarction most common arrhythmia reported was ventricular ectopics (41.1 %) followed by supraventricular ectopics (29.4%). Out of the 47 patients of inferior wall myocardial infarction only 3 patients (6.38%) were having ventricular tachycardia with equal percentage of patients in each group.

(10) Statistically significant relationship between R/Q ratio and mean ejection fraction was seen in the present study. Mean ejection fraction of patients with R/Q ratio> 2 (group 1) was 49.91±5.99, 46.10±5.84 in patients with R/Q ratio 1-2 (group II) and 42.0±6.32 in patients of R/Q ratio < 1 (group III)

(11) Out of the 22 patients in group I (R/Q ratio> 2), 14 patients (63.64 %) were having hypokinesia and 7 patients (31.8%) were having akinesia of inferior segment of LV. Out of the 44 patients of inferior wall myocardial infarction 22 patients were having hypokinesia in which 14 patients (63.64 %) belonging to group I (R/Q ratio> 2) only 2 patients (9.0%) belonged to group ill (R/Q ratio < 1). So present study shows that with good R/Q ratio i.e. > 2 severity of regional wall motion abnormality decreases.

(12) In present study distribution of inferior wall myocardial infarction in. relationship to Killip class was statistically significant. Out of 2 cases of Killip class 1, 17 (53.12%) are from group I (R/Q ratio> 2) and 3 cases (9.37%) are from group III (R/Q ratio <1). Only one case of Killip class IV was reported from the present study, which was from group III (R/Q ratio < 1). Majority of group II (R/Q ratio 1-2) patients are in KiI1ip class I and II (85.5%).

(13) Out of 26 patients of IWMI thrombolyzed, 22 patients had no change in R/Q ratio on 7th day as compared to 3rd day, 4 patients showed a lower R/Q ratio on 7th day. But in non thrombolyzed cases of IWMI 8 patients had a lower R/ Q ratio on 7th day as compared to 3rd day. So lower R/Q ratio on 7^{th} day in nonthrombolized patients as compared to thrombolyzed patients of IWMI was statistically significant. Successful thrombolytic therapy will prevent the extent of inferior wall myocardial infarction.

(14) Out of 22 patients of group I (R/Q ratio> 2) 90% of patients are in group A, none of them had QRS duration> 0.11 sec (group C). But in group III (R/Q ratio < 1) 25% of patients are of group A, and 25% are of group C. As R/Q ratio decreases from > 2 in group I to <1 in group III, percentage of distribution of patients with QRS prolongation (> 0.09 see.) increases and patients with normal QRS duration (< 0.09 see.) decreases. These results are statistically highly significant.

(15) In the present study, out of the 75 patients 6 (8.0%) expired. One from group A (2.0%), 3 from group B (16.67%) and 2 (25.0%) from group C. In the mortality group except one patient of AWMI rest of them expired before echocardiography could be done. So correlation with ejection fraction and R/Q ratio was not possible. Hence mortality was analysed on the basis of QRS duration. Analysis of present study data shows that increased QRS duration had a significant statistical relationship with mortality ($p< 0.05$).

CONCLUSION

The present study shows that the estimation of QRS duration in patients of acute myocardial infarction offer important indirect quantitative information regarding the severity and extent of myocardial infarction. A QRS duration of > 0.09 sec predicts more incidence of arrhythmia, heart failure and other complications. This study shows that both intermediate QRS prolongation (0.09 - 0.11 see.) and significant QRS prolongation (> 0.11 sec) is an eye-opener to worse short term prognosis of myocardial infarction. The current study also concludes that QRS duration < 0.09 seconds is indicative of a relatively benign short term outcome.

The present study also shows that the estimation of the R/Q ratio in lead II of patients with IWMI offers important indirect information regarding the severity and extension of the myocardial damage. An R/Q ratio of > 2 predicts mild localised LV involvement with good global LV function and good clinical course. An R/Q ratio of between 1 and 2 predicts a greater degree of local asynergy and reduced LV function, but still a good clinical outcome. Finally, R/Q ratio of < 1 predicts severe inferior wall asynergy with reduced LV function and complicated clinical course. Current study also indicates that successful thrombolytic therapy will prevent the extent of inferior wall myocardial infarction.

BIBLIOGRAPHY

1. R. Gupta: Epidemiological Evolution and Rise of Coronary Heart Disease in India. South Asian J. Preventive Cardiology, 1997.

2. Janus E.D., Postiglione A, Singh R.B. and Lewis B: The modernization of Asia. Implications for coronary heart disease. Circulation 1996; 96: 2671-2673.

3. Gupta R, Gupta VP: Meta-anlyais of Coronary Heart Disease Prevalence in India, Indian Heart J. 1996; 48: 41-45.

4. Park K: Coronary Heart Disease cited in Parks Text Book of Preventive and Social Medicine(15th Ed. P. 270-271), 1997.

5. Braunwald, Antman: Acute Myocardial Infarction. Cited in Textbook of Cardiovascular Medicine. Ch. 35; 6th Ed. 1114-1205, 2001.

6. Antman EM, Braunwald E, Beasley JW: ACC/ AHA Guidelines for the management of patients with unstable angina. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients .with Unstable Angina).]. Am. Coll Cardiol36: 970-1062. 2000.

7. Swan HJC, Forrester JS, Diamond G: Hemodynamic spectrum of myocardial infarction and cardiogenic shock. Circulation 45: 1097, 1972.

8. Schuster EH, Bulkley BH: Ischemia at a distance after acute myocardial infarction: A cause of early postinfarction angina. Circulation 62: 509-515, 1980.

9. Antman EM, Gugliano RP, Gibson CM et al: Abciximab Facilitates the Rate and Extent of Thrombolysis; Results of the Thrombolysis in Myocardial Infarction (TIMI) 14 Trial. TIMI 14 Investigators. Circulation 99; 2720-2732, 1999.

10. Graner LE, Gershen BJ Orlando MM : Bradycardia and its complications in the prehospital phase of acute myocardial infarction. Am. J. Cardiol 32: 607, 1973.

11. Cohn IN, Guiha NH, Broder MI, Limas CJ: Right ventricular infarction clinical and haemodynamic features. Am. J. CardioloRY: 33; 209-214,1974.

12. Tenenbaum A, Leor J,Motro M. : Improved posterobasal segment function after thrombolysis is associated with decreased incidence of significant mitral regurgitation in a first inferior myocardial infarction. J Am Coll Card iol:25; 1558-63, 1995.

13. .Pedoe-Tunstall H, Kuulasmaa K, Amouyel P : Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Circulation 90: 583-612, 1994.

14. Adams J, Abendschein D, and Jaffe A: Biochemical markers of myocardial injury: Is MB creatine kinase the choice for the 1990s? Circulation: 88; 750, 1993.

15. Ellis AK: Serum protein measurements and the diagnosis of acute myocardial infarction. Circulation 83: 1107-1109, 1991.

16. Walsh BW, Schiff I, Rosner B: Effect of PostMenopausal Estrogen Replacement on the Concentrations and Metabolism on Plasma Lipoproteins. New Eng. J. Med. 375: 1196-1204; 1991.

17. Roberts R, Sobel BE : Creatine kinase isoenzymes in the assessment of heart disease. Am. Heart J. 95: 521, 1978.

18. Haeschane C, Hamm CW, Goldmann B: Troponin concentrations for stratification of patients with acute coronary syndrome in relation to therapeutic efficacy of Tirofiban PRISM Study Investigators. Platelet Receptor Inhibition in Ischemic Syndrome Management. Lancet 354: 1757-1762, 1999.

19. Berger PB and Ryan TJ: Inferior myocardial infarction: High-risk subgroups Clinical progress series. Circulation: Vol. 81; No.2; pp. 401-411, 1990.

20. Carl J. La vie, Bemanrd J. Gersh : Acute myocardial infarction: initial manifestations, Management and prognosis. Mayo Clin Proc. 65: 531-548, 1990.

21. Horan LG, Flowers NC, Johnson J: Significance of the diagnostic Q wave of myocardial infarction. Circulation: 43; 428-36, 1971.

22. Katz LN: Electrocardiography, 2nd Ed. Lea and Febiger, Philadephia, 1946.

23. Marriott JHI: Myocardial infarction. In Marriott JH ed. Practical Echocardiography. Baltimore. Williams and Williams 1972,22653

24. Schamroth Land Schamroth C. : Localization of MyorardiallnfarctIon: In 'An Introduction to Electrocardiography' by Leo Schamroth. 7th Edition, Blackwell Science, Edinburgh, pp. 142-155, 1990.

25. Harpaz D, Behar 5, Gottlieb 5 et al: Atrioventricular and Intraventricular Conduction Disorders in Acute Myocardial Infarction. A Reappraisal in the Thrombolytic Era. Pacing Clin. Electrophysiol21: 2651- 2663, 1999.

26. Blackburn H: Classification Of Electrocardiogram for Population Studies: The Minnesota Code: J Electrocardiol; 2; 305-10, 1969.

27. Haisa Y, Morimoto,S, Wada T: Differentiation between left circumflex and right coronary artery occlusion. Studies on ST segment deviation during percutaneous transluminal coronary angioplasly. Clin. Cardiol. 13; 7H.

28. Velazouez J.R. : Echocardiography assessment of Idl ventricular diastolic hehaviour in aortic stenosis. J. Cardiovaso Ultrasonography, -I Y83; 2: 22Y.

29. Schillcr NB, Shah P.M., Crawford M : Recommendation for quantitation of the left ventricle by two dimensional echocardiography. J. Am. Soc. Echocardiogr... 2; 358, 1989.

30. Henry WL,Kisslo, DeMaria A: Report of the American Society of Echocardiography Committee on Nomenclature and Standard in Two-dimensional Echocardiography. Circulation 1979; 60: 531-38.

31. Henry WL,Kisslo, DeMaria A: Report of the American Society of Echocardiography Committee on Nomenclature and Standard in Two-dimensional Echocardiography. Circulation 1979; 60: 531-38.

32. American Society of Echocardiorgraphy Committee on Standards: Subcommittee on Quantitalion of two-dimensional echocardiography. Recommendations for quantitation of the left ventricle by two dimensional echocardiography. Journal of American Society of Echocardiography. 1989; 2: 5, 358-367.

33. Richard E, Van Reet, Miguel A Quinones, Lawrence R. Poliner: Comparison of two-dimensional echo cardiography with gated radionuclide ventriculography in the evaluation of global and regional left ventricular function in acute myocardial infarction. JACC, Vol. 3, No.2, 243-52, 1984.

34. .Peter Steele, Dennis Kirch: Prompt return to normal of depressed right ventricular ejection fraction in acute inferior infarction. Br. Heart Journal 1977; 39: 1319-1323.

35. Carr KW, Engler RL, Forsythe JR, Johnson AD: Mei)surement of left ventricular ejection fraction by mechanical cross-sectional echocardiography. Circulation 1979; 59, 1196-206.

36. Hochman JS, Sleeper LA, Webb JG: For the Shock Investigators: Cardiogenic. Shock Complicating Acute Myocardial Infarction. Am. J. C. 36: 1063-1070, 2000.

37. Bates ER: Revisiting reperfusion therapy in inferior wall myocardial infarction. J Am Coll Cardiol: 30; 334-42, 1997.

38. Pudil, Micha S, Feinberg: The Prognostic Significance of Intermediate QRS Prolognation in Acute Myocardial Infarction. International Journal of Cardiology 78; 2001, 233-239.

39. Hathaway WR, Peterson ED, Wagner GS, Granger CB: Prognostic Significance of the Initial Electrocardiogram in Patienlc; with Acute Myocardial Infarction. GUSTO-1 Investigators. Global Utilization of Streptokinase and t-P A for Occluded Coronary Arteries. JAMA 279: 387-91, 1998.

40. Murkofski RL, Dangas G, Diamond JA, Mehta D, Schaffer A: A prolonged QRS duration on surface electrocardiogram is a specific indicator of left ventricular dysfunction. J. Am. ColI Cardiol 1998; 32: 476-82.

41. Brembilla Perrot B, Houriez P, Claudon 0: Evolution of QRS duration after myocardial infarction: clinical consequence. PacinR Clin. ElectrophysiollYY99; 22: 1466-1475.

42. Michaelides A, Ryan JM, Vanfossen D: Exercise induced QRS Prolongation in patients with coronary artery disease: a marker of myocardial ischaemia. Am. Heart J. 1993; 126: 1320-1325.

43. Yochai Birnbaum: Prognostic Significance of the Admission ECG in Acute Myocardial Infarction. JACC Vol. 27; 1128-32, 1996.

44. Horwich T, Lee SJ, Saxol: Usefulness of QRS Prolongation in Predicting Risk of Inducible Monomorphic VT in Patients Referred for Electrocardiographic Studies. Am. J. Cardiol 2003, Oct 92(7): 804-9.

45. Pedretti R, Etro M.P., Laporta A, Braga 5.5., Caru B: Prediction of Late Arrhythmic Events aftl~r Acute Myocardial Infarction from Comhined Use of Non-invasive Prognostic Variuhles and Inducibility of Sustained Monomorphic VT. Am. J. Cardiology 1993; 71: 1131-41.

46. Brilakis, Nikolaos C: Usefulness of QRS duration in the absenn~ of bundle branch block as an early predictor of survival in Non-ST Elevation Acute Myocardial Infarction. Am. J. Cardiol 2002; 89: 1013-1018.

47. Fadl YY, Zareba W, Moss AJ: Prolonged QRS Duration and Cardiac Death in Postinfarction Patients. PACE 26: S21 (Abstr) 2003.

48. Schamaroth L: Inferior Wall Myocardial Infarction. The Electrocardiography of Coronary Heart Disease. London: Blackwell Scientific Publication, 1975; 47-50.

49. Page DL, Caulfied JB, Kascor JA: Myocardial Changes Associated with Cardiogenic Shock. N Engl J Med 1971;285:133-37

50. Alexander Arditti, Samuel Sclarovsky, Ruben RF Lewn, Charles Helman, Boris Strarberg, Nili ZafTir and Jacob Agman: A simplified QRS Scoring System for the Estimation of Severity of Acute Inferior Wall Myocardial Infarction. Chest, 87, 6 june1985, 1988; 777-785.

51. Eliezer Klainman, Samuel Sclarovsky: The Nature Course of Electrocardiographic Stages of Acute Inferior Myocardial Infarction in Regard to R/Q Ratio Group Classification. Journal of Electrocardiology, 21 (4), 19HH, 293-301.

52. Lewin Arditti Strasberg: Predominant Right Ventricular Infarction. Clinical and Electrocardiographic Features. European Heart Journal, 1986; 7: 425-430.

53. Mark Cohen, Steven JS, Peter Rentrop: Prospective analysis electrocardiographic variables as markers for extent and location of acute wall motion abnormalities observed during coronary angioplasty in human subjects. J. Am. ColI. Cardiol 1987; 10: 17-24.

54. Eaton LW, Weiss JL, Bulkley BH: Regional cardiac dilalalion after acule myocardial infarction recognition by lwo dimensional echocardiography. New. Eng. J. med. 1979; 300: 57-62.

55. Strass HD, Sobel BE, Roberts R: The inOuenre of right ventricular infarction on enzymatically estimated infarct size. Haemodynamics and prognosis. Circulation 1980; 62: 503-508.

56. Miller RR, Amsterdam EA, BORren HG, Massumi RA, Zclis R, Mason DT: Electrocardiographic and cineangiographic correlations in assessment of the location, nature and extent of abnormal left ventricular segmental contraction in coronary artery disease. Circulation, 1974; 19: 445-54.

57. Ideker RE, Behar VS, Wagner GS: Evaluation of asynergy as an indicator of myocardial fibrosis. Circulation 1978; 57: 715-25.

S.no	Name	Age	Sex	R/U	Rel	Presenting Symptoms					Past History					Per his			General Examination							Resp. Sy
						C.P	Swe.	Nau.	Vom.	Sync.	Breath	Palp.	H.T.	D.M	H.D	Addic			Diet	Ht	Wt	BMI	B.P.		JVP	Basal Crep
																T	S	A					Sys	Dias		
1	B.K	60	M	R	H	3.00AM	+	+	+	-	-	-	+	-	-	-	-	-	V	1.7	50	17.3	60		+	+
2	G.S	55	M	R	H	6.3AM	+	+	+	-	-	-	-	-	-	+	+	-	V	1.68	70	24.8	150	90	-	-
3	S.T	65	F	U	H	12.3PM	-	-	+	-	-	-	-	+	-	-	-	-	V	1.5	56	24.89	110	76	-	+
4	S.D	74	F	R	H	4.00AM	-	-	-	-	-	-	-	+	-	+	-	-	V	1.72	51	17.24	130	80	-	+
5	S.D	40	M	R	H	6.3PM	+	-	-	-	+	-	-	-	-	+	-	-	V	1.6	60	23.44	110	76	-	-
6	R.P	80	M	U	H	7.3AM	+	-	+	-	+	-	-	-	-	-	-	-	V	1.73	74	24.73	60	-	+	+
7	R.L	60	M	U	H	5.00AM	+	-	-	-	-	-	+	-	-	+	-	-	V	1.6	52	20.31	110	70	+	+
8	R.B	45	M	R	H	1.3PM	+	-	-	-	-	+	-	-	-	+	+	-	V	1.67	58	20.8	100	80	+	-
9	G.S	75	M	R	M	7.3PM	+	+	-	-	+	-	-	-	-	+	-	-	V	1.72	72	24.34	140	80	+	+
10	S.M	68	M	R	H	6.00AM	+	-	-	-	-	-	+	-	-	+	+	-	V	1.74	70	23.12	126	80	+	-
11	R.K	61	M	R	H	8.3AM	+	+	-	-	+	-	-	+	-	+	-	-	M	1.5	60	26.67	130	80	+	+
12	A.B	95	M	U	H	2.00AM	+	+	-	-	+	-	-	-	-	+	+	-	V	1.68	74	26.22	84	60	-	+
13	R.B	73	F	U	H	8.3PM	+	-	-	-	-	-	+	-	-	-	+	-	V	1.7	52	17.99	100	70	-	+
14	P.S	42	M	U	H	1.00AM	+	+	+	-	+	-	-	+	-	+	+	-	M	1.78	85	26.83	-	-	-	+
15	M.H	70	M	R	M	9.3AM	+	-	-	-	+	-	-	-	-	+	+	-	V	1.74	72	23.78	120	80	-	+
16	J.P	72	M	R	H	1.3AM	+	-	-	-	-	-	-	-	-	-	-	-	M	1.76	72	23.24	130	86	-	-
17	R.P	72	M	R	H	9.3PM	+	-	-	-	+	-	-	-	-	-	-	-	V	1.86	85	27.28	110	70	-	-
18	J.P	70	M	R	H	10.3AM	+	-	-	-	+	-	-	-	-	+	-	-	V	1.56	68	27.94	110	80	+	-
19	U.P	60	M	R	H	2.3AM	+	-	-	-	-	-	-	-	-	+	-	-	M	1.68	68	24.09	130	70	+	-
20	K.P	50	M	U	H	2.3AM	+	-	-	-	-	-	-	-	-	+	-	-	V	1.62	65	24.77	110	80	+	-
21	D.S	52	M	R	H	3.3AM	+	-	-	-	-	-	-	-	-	+	-	-	M	1.72	76	25.69	100	80	+	-
22	B.L	60	M	U	H	10.3PM	+	-	-	-	-	-	-	-	-	+	-	-	V	1.62	78	29.72	130	90	+	-
23	R.P	82	M	R	H	11.3PM	+	-	-	-	-	-	+	+	-	-	-	-	V	1.68	82	29.05	130	80	-	-
24	R.N	65	M	R	H	4.3AM	+	-	-	-	+	-	-	-	-	+	+	-	V	1.5	62	27.56	80	60	-	+
25	I.K	65	M	U	M	11.3AM	+	-	-	-	-	-	-	+	-	+	+	-	V	1.6	68	26.56	120	80	-	+
26	S.L	65	M	U	H	7.00PM	+	-	-	-	-	-	-	-	-	+	+	-	V	1.61	76	29.3	138	80	-	+
27	I.W	55	F	R	H	5.3AM	+	-	-	-	-	-	-	-	-	+	+	-	V	1.58	60	24.03	110	80	-	+
28	R.G	42	M	U	H	7.00AM	+	-	+	-	-	-	-	-	-	+	+	-	V	1.7	68	23.23	130	80	-	-
29	M.L	65	M	R	H	3.3PM	-	-	-	-	-	-	+	-	-	+	-	-	V	1.75	54	17.63	134	90	-	-
30	R.W	40	M	R	H	7.00AM	+	-	-	-	-	-	-	-	-	+	-	+	V	1.68	78	27.64	120	80	-	-
31	H.K	60	M	R	M	1.00AM	+	-	+	-	-	-	-	+	-	+	-	+	V	1.68	62	21.97	140	70	-	-
32	R.A	65	M	U	H	8.00PM	+	-	-	-	-	-	-	-	-	-	+	-	V	1.56	58	23.83	140	120	-	+
33	K.W	55	F	R	H	2.00AM	-	-	-	-	-	-	-	-	-	+	+	-	M	1.64	60	22.31	100	70	-	+
34	N.M	62	M	R	H	8.00AM	+	-	+	-	-	-	+	+	-	+	+	-	V	1.6	72	28.13	200	80	-	+
35	M.N	50	M	R	H	3.00AM	-	-	+	-	-	-	-	-	-	-	+	-	V	1.7	53	18.34	110	80	-	-
36	D.S	35	M	U	H	3.3PM	+	-	-	-	-	-	-	-	-	+	-	-	V	1.72	66	22.3	140	80	-	-
37	R.J	60	M	R	H	4.00AM	+	-	+	-	-	-	-	-	-	+	+	-	V	1.62	60	22.86	124	80	-	-

'st.
Killip Gr

IV
IV
II
I
I
IV
III
I
II
I
III
III
III
IV
I
I
I
I
I
I
I
I
I
I
III
II
II
I
I
I
I
I
I
II
I
I
II

S.NO.	Investigation					T/t Given		Arrthythmia							E.C.G.			ECHO		Diagnosis	Outcome	
	Hb	TLC	B.S.	B.U.	S.Cho	SK	Dose	Anti.Insch	Ant.Coau.	S.V.E.	V.E.	S.V.T	V.T	C.H.B	R/Q Ratio 3 Day	R/Q Ratio 7Day	QRS Duration	E.F.	Wall Motion			
38	11.5	5600	80	34	190	-		+	+	-	-	-	-	-	3	2.5	0.06	52	HYPO.IW		IWMI	D
39	12	8800	78	32		SK	15	+	+	-	-	-	-	-	4	4	0.08	45	AKINA.IW		IWMI	D
40	11	9900	80	26		-		+	+	-	-	-	-	-	1	0	0.08	44	AKINA.IPW		IWMI	D
41	11.5	10600	116	30		-		+	+	-	-	-	-	-	-		0.1	40	INA.IVS.APEX		AWMI	D
42	8.9	10600	74	32		SK	15	-	-	-	-	-	-	-	-		0.08	48	AKINA.ILW		AWMI	D
43	9.8	9600	112	36	180	-		+	+	-	-	-	-	-	-	4	0.06	44	'PO.IVS.APEX		AWMI	D
44	10.6	9400	74	38	170	-		+	+	-	-	-	-	-	4	3	0.08	40	AKINA.IVW		IWMI	D
45	8.8	10100	118	34	250	SK	15	+	+	-	-	-	-	-	5		0.06	52	HYPO.IVW		IWMI	D
46	9	11600	120	26	190	-		+	+	-	-	-	-	-	-		0.09	45	INA.IVS.APEX		AWMI	D
47	9.4	9800	116	30	180	SK	15	+	+	-	-	-	-	-	1.25	1.25	0.1	46	AKINA.IW		IWMI	D
48	8.2	10200	86	30	230	SK	15	+	+	-	+	-	-	-	2.5	2.5	3.08	48	HYPO.IW		IWMI	D
49	10.2	9600	80	26	180	SK	15	+	+	-	-	-	-	-	1	0	0.12	44	AKINA.ILW		IWMI	D
50	11	10100	78	34	180	-		+	+	-	-	-	-	-	2	2	0.08	54	AKINA.IW		IWMI	D
51	9.8	9600	120	30	210	-		+	+	-	+	-	-	-	1	1	0.08	54	AKINA.IW		IWMI	D
52	10.6	9400	122	32	212	-		-	+	-	-	-	-	-	1	0	0.08	36	KINA.IW,IVS		IWMI	D
53	9.8	9600	116	34	170	SK	15	+	+	-	+	-	-	-	1	1	0.08	49	HYPO.IW		IWMI	D
54	10.6	9400	70	30	190	SK	15	+	+	-	+	-	-	-	-		0.08	48	AKIN.IVS		AWMI	D
55	11	9500	76	32	240	SK	15	+	+	-	-	-	VT	-	-		0.1	32	.IVS.APEX.ALW		AWMI	D
56	8	10600	116	32	150	SK	15	+	+	-	+	-	VT	-	-		0.08	45	.IVS.APEX.ALW		AWMI	D
57	8	9800	78	32	240	SK	15	+	+	-	-	-	-	-	-		0.06	50	.IVS.APEX.ALW		AWMI	D
58	8	10600	72	3	220	SK	15	+	+	-	+	-	-	-	-		0.08	44	.IVS.APEX.ALW		AWMI	D
59	11	9860	80	32	180	SK	15	+	+	-	-	-	-	-	3	3	0.08	48	AKIN.IW		IWMI	D
60	10.1	5600	78	28	110	-		+	+	-	+	-	-	-	-		0.1	40	.IVS.APEX.ALW		AWMI	D
61	10.6	9400	80	30	230	SK	15	+	+	-	-	-	-	-	2.5	2.5	0.06	50	AKIN.IW		IWMI	D
62	11	9500	116	28	250	SK	15	+	+	-	+	-	-	-	4	4	0.06	50	HYPO.IW		IWMI	D
63	8	10600	74	30	190	SK	15	+	+	-	-	-	AIVR	-	3	3	0.08	50	AKIN.IW		IWMI	D
64	8	9800	112	28	255	SK	15	+	+	-	-	-	-	-	-		0.08	41	.IVS.APEX.ALW		AWMI	D
65	8	10600	116	40	235	SK	15	+	+	-	+	-	-	-	-		0.06	48	.IVS.APEX.ALW		AWMI	D
66	11	9860	70	36	225	-		+	+	-	+	-	-	-	-		0.12	40	AKIN.IVS		AWMI	D
67	10.1	8600	76	28	170	SK	15	+	+	-	-	-	-	-	-		0.1	44	.IVS.APEX.ALW		AWMI	D
68	10.6	9400	116	30	215	SK	15	+	+	-	+	-	AVIR	-	4	4	0.1	38	AKIN.IW.IVS		IWMI+AWM	D
69	11	9500	78	28	190	-		+	+	-	-	-	VT	-	2	2	0.08	48	HYPO.IW		IWMI	D
70	8	10600	72	34	250	SK	15	+	+	-	+	-	-	-	3	3	0.06	60	NORMAL		IWMI	D
71	8	9800	80	26	180	SK	15	+	+	-	-	-	-	-	0	0	0.08	38	AKIN.IPW		IWMI	D
72	8	10600	78	40	220	-		+	+	-	+	-	-	-	-		0.1	40	.IVS.APEX.ALW		AWMI	D
73	11	9860	80	36	170	-		+	+	-	+	-	-	-	-		0.08	42	.IVS.APEX.ALW		AWMI	D
74	10.1	8600	116	34	210	-		+	+	-	+	-	-	-	-		0.06	70	NORMAL		AWMI	D
75	12	7500	74	22	180	SK	15	+	+	-	-	-	VT	-	-		0.1	42	.IVS.APEX.ALW		AWMI	D

Department Of Medicine
Government Royapettah Hospital, Kilpauk Medical College, Chennai-10
PROFORMA
"STUDY OF QRS DURATIONAND R/Q RATIO IN THE ASSESSMENT OF SEVERITY OF ACUTE MYOCARDIALINFARCTION"

Case No.: IP No.: DOA: DOD/E:
Name: Age: Sex:M/F Religion H/M/C
Domicile: R/U
Address
Socio-economic Status
PRESENTING SYMPTOMS
Chest Pain Time of onset Duration Hrs. Mts.
Site Central / Epigastric / Left Sided / Back
Character Burning / Crushing / Gripping / Squeezing / Tightening
Severity Mild / Moderate / Severe
Precipitating or Aggravating Factors:
Relieving Factors:
Other Symptoms
Sweating: Nausea: Vomiting Syncope
Breathlessness Palpitation Oedema Abd. Distension
PAST HISTORY
Hypertension D.M. I.H.D. Others
FAMILY HISTORY

	Diabetes Mellitus	HTN	CAD	Others
Father				
Mother				
Brother				
Sister				

PERSONAL HISTORY
Addiction Duration Quantity
Tobacco Chewing
Smoking
Alcohol
Diet- Veg / Non-Veg / Mixed
GENERAL EXAMINATION
Built: Thin/Normal/ Obese Wt. (Kg): Ht. (Cm) BMI

Pulse	R	Ry	V	Ch	Syn.	Vessel Wall	Peripheral Pulse	RFD

JVP	Ht. (Cm.)	A	X	C	V	Y	Others

B.P. Temp. Pallor Cyanosis

Icterus Oedema Xanthoma/Xanthelasma

SYSTEMIC EXAMINATION

CVS Inspection:

Shape Apex beat

Other Pulsation

Palpation:

Apex beat: Thrill: LPH

Epigastric Pulsation Palpable P2:

Auscultation

	S1	S2	S3	S4	Murmur		Other Sounds
					Systolic	Diastolic	
Mitral							
Tricuspid							
Pulmonary							
Aortic							

RESP. SYSTEM. Basal Crepts

ABDOMEN

Liver	Size	Tender	Consistency	Pulsation

CNS

INVESTIGATION

Hb % TLC /CM.3 DLC P L M E Sugar/AIB

BI.Sugar BI.Urea S. Cholestral Urine R/M

S. Creatinine

TREATMENT GIVEN

Thrombolytic(SK) Y/N Dose:

Anti Ischeamic Nitrates/B Blocker/CBB

Antiplatelet Aspirin/Clopidogrel

Anticoagulants LMWH/Heparin Dose:

ACE Dose

STATINS Dose

ECG FINDINGS

	On Admission	24 Hrs.	48 Hrs.	72 Hrs.	5^{th} Day	7^{th} Day	9^{th} Day
Rate							
Rhythm							
P Wave							
PR Interval							
Width							
Axis							

Q Wave							
Q Wave							
R Wave							
S Wave							
QT							
QTc Interval							
R/Q ratio in Lead-II							
Maximum QRS duration							

ECHOCARDIOGRAPHY

LVEF

Hypokinesia/ Akinesia/ Normal

Day	1	2	3	4	5	6	7	8	9	10
Arrhythmias										
LVF										
Echo.										
Other Events										

Signature of Co-guide

Signature of Candidate

GENERAL EXAMINATION

Built: Thin/Normal/Obese Wt.(Kg): Ht.(Cm.) BMI

Pulse	R	Ry	V	Ch	Syn.	Vessel Wall	Peripheral pulse	RFD

JVP	Ht.(Cm.)	A	X	C	V	y	Others

B.P. Temp. Pallor Cyanosis

Lcterus Oedema Xanthoma/Xanthelasma

SYSTEMIC EXAMINATION

CVS Inspection:

Shape Apex Beat
Other Pulsation

Palpation:

Apex beat: Thrill: LPH
Epigastric Pulsation Palpable P

Auscultation:

	S1	S2	S3	S4	Murmur		Other Sounds
Mitral					Systolic		
Tricuspid							
Pulmonary							
Aortic							

RESP SYSTEM. Basal Creps

ABDOMEN

Liver	Size	Tender	Consistency	Pulsation